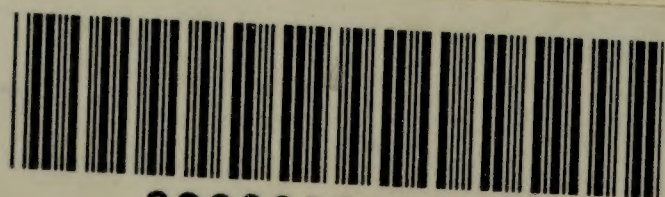






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DISEASES  
OF  
INFANTS AND CHILDREN.







THE  
DISEASES  
OF  
INFANTS AND CHILDREN.

BY  
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## PREFACE.

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THIS work is based upon many years' practical experience of the diseases of early life, the examination of a large number of children, and the necessarily somewhat extensive study of the literature of the subject. Its object is to describe the ailments of children in a form suitable for the general practitioner of medicine and sufficiently detailed to render further reference to other works unnecessary, except in the case of unusually rare diseases.

A liberal amount of space is devoted to surgical affections which may come under the notice of physicians in early stages and under the care of practitioners at all periods of their course. The knowledge of these diseases must be extensive enough to enable the practitioner to clearly understand the nature of the illness, to appreciate the best lines of treatment, and to recognise the conditions for which the aid of a surgeon should be invoked. Indeed, a year's experience as house surgeon is almost essential to the training of a physician.

Considerable attention is given to maladies which have only been fully recognised in recent years, for instance,



## *Preface.*

hypertrophic stenosis of the pylorus in infants, acidosis, delayed anæsthetic poisoning, and cleido-cranial dysostosis. In connection with the disorders of bone formation a new system of nomenclature is suggested. The descriptions of some common ailments have been curtailed, inasmuch as in a book for graduates it is unnecessary to amplify the minutiae of symptoms, diagnosis and treatment. In discussing the various therapeutic measures no special method is dogmatically insisted on, for it is obvious that remedies applicable in one case may be contra-indicated in another by the character of the illness, the idiosyncrasy of the child, or the social position and environment. In children, even more than in adults, we have to deal with a patient and relatives, often a nurse as well, in addition to treating the particular disease.

Illustrations and charts have been omitted. The kindergarten teaching of medicine by pictures, diagrams and models, is advantageous for the student, but may prove a disadvantage to those in active practice. Photographs and drawings, except of pathological states, are of little value, unless so typical that the veriest tyro can recognise the disease from a description. They are injurious in that the expectation of seeing the typical cases of pictorial



## *Preface.*

illustration increases the danger of overlooking those early stages in which disease is amenable to treatment.

My indebtedness to the valuable contributions of other writers is obviously very great, and must be warmly acknowledged. That continual reference in the text to individuals is wanting is not due to any desire to claim undeserved credit, but simply in order not to burden the work with innumerable names and references. Perhaps at times statements and opinions have been put forward in a somewhat dogmatic manner. If so, the excuse is that they are the outcome of prolonged thought and considerable experience.

The Society for the Study of Disease in Children was formed in 1900, and after eight years' active work became amalgamated with the Royal Society of Medicine. In 1904 the late Dr. George Carpenter founded the *British Journal of Children's Diseases*. The study of disease in early life has consequently received such a great impetus in this country that, in my opinion, a new systematic and up-to-date book on the subject is required.

EDMUND CAUTLEY.

LONDON, W.,  
*April, 1910.*







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# CHAPTER I.

## INTRODUCTORY.

*Definitions—The Examination of Children—Special Features of Disease in Children—Growth and Development.*

The field of study of disease in children covers in some respects a more extensive area than in the case of adults, while in others its boundaries are more strictly defined. Inflammatory and infective disorders are widely distributed and more virulent in action, and the affections dependent on an endogenous failure of nutrition, or abiotrophy, show themselves most frequently in early life. Disease is less influenced by the prolongation of injurious habits of life and the degenerative affections due to age, changes in the organs or environment. Pathological processes occur in their simplest forms, and are less complicated.

The child is the father of the man, but is by no means a miniature adult. Although there are comparatively few special diseases in children, the manifestations of disease in them, and to a greater extent in infants, differ much from what is seen in adults. Diagnosis is not necessarily more difficult, and in one way is easier, for examination can be, and has to be, conducted more thoroughly. There is, consequently, less likelihood of overlooking important facts. An adult is apt to direct attention to one particular symptom or organ, so the remainder may escape examination. The interpretation of nervous symptoms in particular is difficult. Always there is a liability to misinterpret facts from lack of acquaintance with the peculiarities of illness in early life.

A thorough knowledge of disease at this period implies a wider appreciation of the problems of life and pathology than is necessary for those whose work is mainly among adults. In the one case we have to deal with life in the making, in the other with the finished product, by no means a perfect work of art. Most affections of later life are seen at some time or other in children and, in addition, a certain number incompatible with prolongation of life.

It is essential that a special knowledge of diseases in early life should be accompanied by a sound practical acquaintance with those of later life, for the child grows into the adult, and the life of the adult constantly depends on the proper management of the child in its earliest years.



**Definitions.**—The period of infancy terminates at the completion of the first dentition, roughly about the middle of the third year of life. This can be subdivided into three different stages. During the first week the child may be spoken of as the “Newborn.” Strictly speaking, a newborn child enters the world at the end of the fortieth week of pregnancy. Born before that date it is spoken of as premature, and according to the degree of prematurity it requires special treatment. It passes from an equable temperature and a watery bed into a world where the temperature is variable and exposure to injury constantly liable to occur. The change is enormous. During the first fortnight the newborn may suffer from effects of the confinement, and these effects may be prolonged or even permanent. Thus, it may be born asphyxiated, due to strangulation by the cord; or the lungs may be unexpanded (atelectasis); or it may have a cephal-hæmatoma or tumour of the sterno-mastoid; or it may exhibit one or other of the birth palsies; or suffer with ophthalmia or from septic infection. During this period the alimentation of the child undergoes an abrupt change. From dependence for food on the maternal blood supply, it suddenly is compelled to live upon a different kind of food introduced into an untrained stomach. Bad feeding, especially the giving of artificial foods during the first few days of life, may upset digestion and assimilation to such an extent as to prove fatal.

Up to the end of the ninth month the child may be spoken of as the “Suckling” or the “Baby”; the term “Infant” being reserved for the subsequent period up to the end of the middle of the third year. Some writers use the term *première enfance* to mean the period before the eruption of the milk teeth begins, and others use it to denote the whole of the first year. During this period life is mainly vegetative, and the reflex functions predominate. This is especially so during the first few months. Growth is very rapid, and great strain is thrown on digestion and metabolism. The result is that many of the affections of the first year are due to dietetic errors, which lead to disorders of digestion, marasmus, scurvy, and rickets.

It is during later infancy, after the suckling period, that the effect of bad feeding on the osseous and muscular systems is most marked; the deformities due to rickets developing when the child begins to crawl, stand, and walk.

The name “Childhood,” or *seconde enfance*, is sometimes used for the period extending from the commencement of the first to that of the second dentition. It is preferable to use the term “Early Childhood” to cover the period from the end of the first to the commencement of the second dentition. During this stage growth is very active, and the dependence on digestion and assimilation very great.

“Late Childhood,” or *Youth*, extends from the onset of the second dentition up to puberty. During this period there are comparatively few



affections peculiar to children, beyond those dependent upon undue strain on the nervous system by education.

“ Adolescence ” commences with puberty, at about twelve to fourteen years of age, and ends about the eighteenth year.

**The Examination of Children.**—It is of little value spending much time on the details of the mode of examination of the very young. There are many methods equally satisfactory in the hands of those who adopt them. Examination must be much less prolonged than in adults, and great reliance has to be placed on objective signs. An important point to remember is that a child is a young animal, nervous, on the watch, and very easily startled or frightened. Anyone who, unaided, has succeeded in haltering a foal for the first time, will understand how to undertake the examination of a child. The chief difficulties arise from the liability to fright, crying, and struggling, and from the inability to describe and localize pain and other symptoms. Counterbalancing advantages are the ease with which the child can be stripped and completely overhauled, the ease with which it can be moved about during examination, and the absence of prevarication.

A child is distrustful of strangers, still more so if the doctor has been held up as a bogey, instead of the child's best friend, or if a previous visit to a dentist has caused the infliction of pain. Never look a baby in the eyes, although inspection is of chief importance. Children, even babies, like animals, intensely dislike being stared at. On entering the sick room, or if the child is brought into the consulting room, it is rarely advisable to try at once to make friends. First talk to the mother or nurse and learn the history of the present and previous illnesses, the number of children, and where the patient comes in the family; the number and health of those living; the causes of death of any which are dead; the mother's health during pregnancy; the nature of the confinement, and of any difficulties or abnormalities at birth; the number of miscarriages, and the mode of feeding. Inquire as to a history of asphyxia or fits at birth, the weight, the rate of growth, the date of teething, sitting up, walking, talking, etc. In the history of past ailments, inquire more particularly as to snuffles, rashes, bronchitis, vomiting and diarrhœa, infectious disease, and ear and throat troubles. It is much better to make such inquiries before investigating the particular illness, or the parent may assume that you are trying to ascribe its causation to something in the family or past history. Ready as a parent may be to transmit disease to the child, there is a natural objection to taking the blame thereof, and it may be both inadvisable and unnecessarily unkind even to suggest its possibility. Heredity is, however, an important factor in so many diseases that the family history should not be neglected. The terms used must be analysed, for such expressions as decline, consumption of the lungs or bowels, nervous disease, etc., have varied meanings.



It is equally important to pay serious attention to the history of the illness given by the mother or attendant, noting especially what has led to medical advice being sought, even though it involves delay, repetition, and garrulity. The baby cannot talk, a child may be able to, but is quite incompetent to describe symptoms or to locate pain with trustworthy accuracy. An observant mother or nurse can give valuable information, which must be carefully weighed, for stress is constantly laid on the trivial features to the neglect of the serious points in the case. Much importance should be attached to her opinion of the progress of the illness. Ascertain the date of onset, or when the child last seemed quite well, and the state of appetite, digestion, sleep, irritability, languor, and debility. Personally examine urine and fæces, and do not trust even a trained nurse's description. The diseases of infancy are comparable to those of animals, for in each case we are dependent on the observation of onlookers, and on physical examination, and are neither assisted nor deceived by subjective symptoms. The character of the cry may indicate discomfort, pain, hunger, indigestion, weakness, temper, or it may be due to mere habit.

It is of great assistance if the child is stripped and wrapped in a blanket, or is in bed before the doctor enters the room, for the removal of the clothes at an unusual time and in the presence of a stranger often leads to distress and temper in even good children. No examination is complete without stripping. If the child is not too ill, examine it on the nurse's knee rather than in bed, for the child feels safer in a sitting posture. While in the room and obtaining the history the doctor should not sit close to and stare at the child, but by occasional glances can acquire some idea of its expression and general appearance, and can say a few words to it or indulge in trivial play. With gentleness and tact it is almost always possible to obtain the confidence of any child, provided all movements are unhurried, patience is unlimited, and the cerebral state is normal.

Note the size and shape of the head, the degree of interest in surroundings and toys, and the facial expression when not masked by emotion. It indicates how the child is feeling and is expressive, for the furrows of age are absent. The facies of disease is only learnt by observation and much practice. Facial diagnosis is of assistance, but must not alone be relied upon. To the expert the location of the disease in the cranial cavity, respiratory organs, heart or abdomen is often clearly written on the face. Thus the head may be retracted in simple meningitis, the expression dull and vacant in tuberculous meningitis, the face drawn and lined in abdominal disease, the mouth open in the presence of adenoids, and so on. The appearance of impending death is indicated by the *Facies Hippocratica*, and that of acute diarrhoea or acute nephritis is also characteristic. On simple observation one may note the flushed face and bright eyes of fever; hurried respiration, dusky complexion and blue lips of pulmonary and some cardiac affections; or that the hand is frequently raised to the head



or ear, because of headache or earache. Simple inspection affords information as to the mental condition of the child, cretinism, infantile myxœdema, idiocy, mongolism, or congenital syphilis. Note the presence of squint, the state of the pupils and eyes, the response to light, and mucus on the cornea ; nasal discharge ; state of the tongue and mouth.

Normal peculiarities of configuration must not be mistaken for signs of disease. Up to the age of three years the head is relatively large. For many years the abdomen is prominent and the chest is round, not acquiring the oval shape for some time. Malformations of the chest, a bulging precordium, and venous dilatation may be obvious on inspection.

If the child is asleep, note its posture, facial expression, character of the sleep, and count the pulse and respirations. The pulse is often irregular during sleep. While awake it is of comparatively little value as a sign of disease, unless it is abnormally infrequent, and intermittent or irregular. Blood pressure is low. Breathing is often strange to the inexperienced. A child may hold its breath for an extraordinary long time, or there may be inversion of the respiratory rhythm. The rate of breathing may be counted if the child is not excited, and is often very rapid in fever. Retraction of the epigastrium and lower ribs indicates respiratory obstruction. Puerile breathing may be mistaken for bronchial breathing, unless the two sides are compared, and bronchial breathing is normally heard in certain situations. It is quite a common mistake to diagnose bronchial breathing on one side, when the condition is really one of exaggerated puerile breathing, with enfeebled breath sounds on the opposite side. Râles are liable to be mistaken for friction, and abnormal sounds are readily transmitted to the opposite side.

Percussion must be light for heavy percussion hurts the child and affords inaccurate information, because of the conductivity of the chest. Percuss in the same phase of respiration or one side may sound less resonant than the other. Dulness over the manubrium may indicate a large thymus or glands. The yielding chest wall readily permits the production of crack-pot sound when the glottis is closed, but it must not be mistaken for that due to a cavity in the lung. Collapse of the lung gives rise to dulness. It is easily produced by obstruction from secretions, and may equally easily disappear. It is constantly mistaken for pneumonic consolidation.

During the general examination of the body it is important to note the state of nutrition, of flabbiness, undue fatness, anæmia, and the presence of rickets or rashes. Palpation should be conducted with a warm hand. It is well borne by babies for they are accustomed to being handled. Begin with the head and note the state of the fontanelles, sutures, bossing, and craniotabes. Examine the neck for enlarged glands. Note whether the skin is unduly hot, dry, moist, or has lost elasticity. Examine the chest for beading of the ribs, and take special care to locate the apex of the heart, the presence of any abnormal pulsation to the right of the sternum



or in the epigastrium, and note the force of the cardiac impulse and the presence of thrills. Note whether there is any respiratory fremitus. Sometimes coarse friction can be felt. Gentle percussion may be then tried, but should be postponed until after auscultation if the child is inclined to cry. Be sure that the child is sitting straight, and that the sound is not deadened by any support.

For auscultation use a binaural stethoscope with a short vulcanite reversible chest piece of different sizes at either end. A soft rubber cover on the mouthpiece is useful to secure proper approximation to the chest wall in thin or wasted infants. A wooden stethoscope is often preferable, but it must be carefully held in the hand so that there is little pressure on the chest wall. If a metal stethoscope is used the end must be warm. Direct auscultation of the back is often less alarming and more satisfactory. Occasionally it is useful for the front of the chest. The best position for the examination of the front of the chest is while the child is sitting up or on the back. For examining the back it should sit up or rest with the face over the nurse's shoulder. A child is rarely frightened of the stethoscope if allowed to play with it first, if the chest piece is warm, and if no undue pressure is exerted. It is generally best to listen to the back first for evidence of fluid and crepitations. In delicate and wasted infants, and in collapse, it is sometimes necessary to make the child cry in order to get it to breathe sufficiently deeply. Without such a precaution patches of collapse and consolidation, and intrapulmonary crepitations may be missed. Listen to the heart sounds and remember that up to the fourth year the pulmonary second sound is normally accentuated.

While examining the chest in a sitting posture the temperature can be taken in the groin. Palpate the abdomen in a sitting posture, and at first while covered up, and examine for enlargement of the liver or spleen, normal kidneys, and abnormal glands. The softness and smallness of the body lends itself to full examination by this method. Note specially the condition of the navel, and the presence of an adherent prepuce or undescended testes. Examine the limbs and note whether they are moved normally, are paralysed, or tender. Test the reflexes. Finally, look at the lips, tongue, teeth, gums, palate, and throat. To make an unwilling child open the mouth, hold the nostrils closed and press the upper lip over the front teeth, or the cheek between the gums, or introduce the handle of a teaspoon to the back of the throat until it retches.

**Special Features of Disease in Children.**—The nervous system is abnormally irritable and susceptible to irritation, reflex and direct. General convulsions and local spasms, e.g., laryngospasm and carpo-pedal contractions, depend upon this. Convulsions are often said to take the place of the rigors which occur in adults at the onset of many diseases. Occasionally this is so, but more commonly acute diseases are ushered in by vomiting. Croupous pneumonia may be mistaken for meningitis when the nervous



phenomena markedly predominate and the signs of consolidation are delayed. Functional nervous derangement is common and especially apt to occur in toxæmic cases, with severe and even fatal symptoms, though nothing abnormal is found post mortem.

The temperature rises easily, and perhaps to a height which may be alarming. It should be taken in the groin or rectum, and normally ranges from 98-99.5° F. in babies. The younger the child the more unstable is the temperature for the heat-regulating centre is incompletely developed or imperfectly controlled. A rise of temperature is not as proportionately serious as in the adult, nor a reliable measure of the severity of the exciting cause. More importance must be attached to the general symptoms. It may even be due to hot bottles, or too many blankets, and insufficient ventilation. The pulse and respiration rates are disproportionately accelerated by any pyrexia.

A remarkable absence of pain or complaint of pain is often noticeable in acute tonsillar affections and diphtheria. The generalisation of disease is another characteristic. A local tuberculosis is a most uncommon cause of death. Miliary tuberculosis is frequent in infancy, and comparatively rare in adults. Affections of the mucous membranes are widely distributed, for instance, enteritis associated with broncho-pneumonia.

Some diseases are almost peculiar to children. Many of these are incompatible with life, such as severe forms of congenital heart disease and amaurotic family idiocy. Others rarely occur except in children, viz., rickets, broncho-pneumonia, infantile paralysis, chorea, and, less notably, specific fevers. Some affections, e.g., achondroplasia and mongolism are present at birth and persist in adults. Many diseases are common to both children and adults, but are usually regarded and described from their course in adults, e.g., acute rheumatism and typhoid fever. Congenital cardiac bruits are common, but endocarditis is rare before the age of two years. Hæmic murmurs may be present in earliest infancy. The alimentary and respiratory systems and the brain are most liable to disease.

From another point of view, that of therapeutics, the child is a more satisfactory subject than the adult. It has great reparative powers. It is growing, going up, not down the hill of life. Its tissues are likely to be healthier, and to respond better to treatment. A child does not overwork, has few mental worries, no responsibilities, and can be efficiently guarded, nursed, and controlled. Even in the matter of transference from place to place, the advantage is on the child's side, for it can be easily wrapped up, carried about, protected from cold and draughts, and not subjected to anxiety or fatigue.

**Growth and Development.**—Excluding ante-natal conditions, and those arising from the act of birth, the health and development of the child depend upon its environment. Under this are included those factors



which directly or indirectly influence and modify physical, mental, and moral growth. These factors are hygienic, i.e., all that pertains to air, exercise, washing, clothing, dwellings, and drainage; dietetic; and educational, including amusements. It is by these means that the physician mainly influences the child during health and disease. Their relative importance varies at different periods of childhood.

Certain anatomical peculiarities depend on the necessities of birth, the passage through a narrow canal, or on physiological causes. The cord soon shrivels after being tied; and the umbilical vein is represented by a thin, flat, yellow band with the two dark tortuous umbilical arteries winding round it. Around the base is a reddened areola, where separation shortly takes place. At birth the liver is large, extending from one-half to one inch below the costal margin, and high up in the chest. The bladder is usually distended with urine, and presents a dull area above the pubes.

The head measures 13-14 inches in circumference, round the forehead and occipital prominence. At 3 months it measures 15 inches, and it increases by 1 inch in circumference in each successive 3 months up to 18 inches at 1 year of age. At 5 years it measures 20 inches. The anterior fontanelle is on a level with the surrounding bones, or a little depressed at first. It is diamond-shaped, and varies in size, being about  $\frac{1}{2}$  in. wide and  $\frac{3}{4}$  in. long. The posterior fontanelle is small, triangular, and at birth overlapped by the bones. In rare instances the fontanelles and sutures are ossified at birth, and interfere with labour.

The face is remarkably small in comparison with the cranium. The nose, like that of a monkey, has no bridge to it. The naso-pharynx and posterior nares are small, so that slight causes may produce dangerous obstruction. The Eustachian tube runs nearly horizontally. The external auditory meatus is placed more anteriorly than in the adult. The jaws are small and rudimentary. The gums are composed of fibrous tissue, covered by mucous membrane, and do not meet.

The head and face may be extraordinarily misshapen from pressure at birth. The cranium may be elongated, flattened, and bulge unequally, but the normal shape is gradually acquired as the brain and skull grow. Deformity is rarely permanent, though a certain degree of asymmetry is constant.

At birth the child is covered with a secretion of sebaceous matter, called the *vernix caseosa*. This may even be absent or vary in amount, up to a covering so thick and impenetrable as to prove fatal. It is washed off gently in the first bath, leaving the skin of a healthy brick-red colour. The head looks unduly large, may be bald or covered with fair hair, and occasionally dark hair, an inch or two in length. The eyes are dull, half open and expressionless. The abdomen is prominent on account of the large size of the liver. The limbs are smooth and rounded, the arms being better developed and more muscular than the legs. The grasp of the



hands is strong, and the child can usually support its weight by its grasp on the two fingers.

Certain milestones on the road of growth and development should be noted as indications of normal childhood. The fontanelle is closed at 18-24 months of age, often earlier. Early closure may occur as a sign of microcephaly. Delay may indicate rickets, hydrocephalus, or cleidocranial dysostosis. The order of eruption of the teeth will be discussed later.

Sucking is an instinctive reflex, and so is the grasp of the hands in the newborn. Lack of this instinctive grasp indicates mental or physical defect. The earliest muscular movements are spontaneous or reflex, aimless movements of the eyes, head, face, neck, and limbs. As a rough guide it may be stated that the child can hold its head up at 3-4 months, and makes efforts to grasp at objects in an inco-ordinate manner. It can sit up at 6-9 months, crawl and stand at 9-12 months, walk at 12-18 months, and talk at 18-24 months of age. Children vary much in the mode of acquirement of these various functions. The majority crawl on hands and knees or feet before learning to walk. A few never crawl, but propel themselves rapidly about the floor on the buttocks. At first the child raises itself by grasping some object with the hands, and is able to stand with the aid of the hands and arms. In the next stage it is able to stand unassisted. Then for a month to 6 weeks it stumbles about with an uncertain, ataxic gait, using all available support. In the next stage, lasting 2 or 3 weeks, it tries to walk without supports, sways the trunk from side to side and waves its arms wildly about in order to maintain its balance, just like a person learning to skate. Painful falls at this period may frighten the child and delay its progress.

All these functions vary much in their time of development, partly according to family peculiarities, partly in accordance with the weight of the child and the mental activity. Premature and marasmic infants are backward. Marked delay often leads to medical advice being sought. In some cases the power has not been acquired, in others it has been lost although once developed. It is chiefly for delay in learning to walk, or because the child has "gone off its legs," that the physician is called in. Hypertonicity of the muscles is common in the very young, and may suggest spastic diplegia. It disappears.

Inability to support the head at a proper age is very suggestive of mental defect or cretinism. Hypotonia occurs in imbeciles, especially mongols, and also in rickets. A child unable to walk at 18 months is generally rachitic, paralysed, or idiotic.

Fat, heavy children should never be encouraged to stand or walk. It is even advisable that they should be discouraged, especially if there is the least indication of rickets. If not, bending of the bones is apt to ensue. A child who has not learnt to walk, should be examined for affections of supporting structures and the neuro-muscular system. The defect may



be due to the loss of muscular tone and lax ligaments of rickets ; congenital dislocation of the hips ; myatonia congenita, a condition of idiopathic muscular flaccidity in which the knee jerks are present ; cerebral or spinal paralysis, mental defects, spina bifida, poliomyelitis, post-pertussal hæmatomyelia or cretinism.

The child that has " gone off its legs " must be examined for evidence of infantile paralysis, scurvy, hip disease, rheumatism, epiphysitis or osteomyelitis. Often the loss of function is due to malnutrition or weakness, the result of acute illness, the art having been forgotten. Sometimes the loss of function is associated with an apparent paralysis due to pain. The epiphysitis of congenital syphilis occurs so early in life, usually a few weeks after birth and rarely after six months, that it does not affect these functions to a very conspicuous extent, but the immobility of the limb is found if looked for.

The circumference of the thorax is about equal to that of the head in the second year of life, and should exceed it in the third year. Lack of development of the chest may mean lung mischief.

**Weight.**—Increase in weight is a good indication of satisfactory progress. A baby should be weighed daily during the first month, and once or twice a week in the succeeding 9 months. The same scales should be used on each occasion, such as those made by Messrs. Avery, or an elongated basket tray supported on a recording dial and weighing up to 28lbs. by ounces. The absence of gain in weight or an actual loss in weight may indicate defects in the food supply, some disturbing factor, such as teething, rapid growth, or the onset of some morbid process. The most common causes during infancy are dietetic defects, insufficient clothing, cold weather, teething, dyspepsia, and diarrhœa. Increase in weight should be accompanied by progressive increase in height. Apart from this it may be due to the mere deposition of fat, as in a rickety infant fed on condensed milk. Occasionally it is due to dropsical effusions or simple œdema. A temporary gain in weight may occur in hospital patients although suffering from serious disease, because of the better quality of the food, regular feeding, warmth and nursing. The weekly weighing should take place on a fixed day, such as Sunday, and at the same hour of the day. The weight may be as much as 5-7 oz. less in the morning than in the evening in an infant of 4-6 months of age, because more food is taken during the day time. The child should be washed, wrapped in a warm flannel and then weighed, the weight of the flannel being subtracted subsequently. Suitable charts for recording the weight on can be obtained from Messrs. J. & A. Churchill.

The weight of a child at birth varies considerably, 3,000 grammes or 6½ lbs. may be taken as a fair average, but it is often exceeded, and anything from 6-9 lbs. may be regarded as normal. Boys are generally heavier than girls, and firstborns smaller than other children. The largest



babies are generally those of women from 25-30 years of age. Giant foetuses have been reported weighing from 14 up to  $24\frac{1}{2}$  lbs. They are born dead. The size of the normal baby depends chiefly on the physical health and build of the mother; her food supply, amount of rest, and freedom from worry during pregnancy; and the period of gestation. A limited diet at this period diminishes the size of the child, lessens its vitality, and makes labour more easy.

During the first 3 days of life weight is lost on account of the passage of urine and meconium, evaporation of water from the skin and lungs, and lack of food. The greatest loss takes place in the first 24 hours. The heavier the babe, the greater is the loss. Boys lose less than girls. The child of a primipara is more likely to lose a greater percentage of its original weight, and recover it less quickly than the child of a multipara, because the milk supply is not so quickly established. The drying up and separation of the cord accounts for a loss of about  $\frac{2}{3}$ -1 oz. The total loss varies from 5-10 per cent. of the original weight, from 3-12 oz., generally averaging 7-8 oz., and it is often not regained until the end of the second week. The initial loss can be prevented by utilising a wet nurse or artificial feeding, but there is no special advantage in such measures unless the infant is premature and weak.

Subsequently weight may be regained with remarkable regularity or by fits and starts. Periods of retardation are followed by periods of acceleration in both breast-fed and artificially fed babies. Speaking generally, the initial weight is doubled in 5 months, and trebled in 12-15 months; such results are often exceeded. The following table in a simple form fairly well represents the rate of gain :—

Birth	..	..	..	7 lbs.
5 months	..	..	..	14 lbs.
15 months	..	..	..	21 lbs.
6 years	..	..	..	42 lbs.
14 years	..	..	..	84 lbs.

All such figures and measurements are merely averages, sufficiently near to afford evidence as to whether the child is developing at a more or less normal rate. The rate of gain is more or less proportionate to the initial weight of the infant. Some abnormally small infants gain weight much more rapidly than those who are very much larger at birth. During the first 3 months of life the child gains 5-7 oz. per week; during the second 3 months, 4-6 oz.; and during the third 3 months, 3-5 oz. Sunlight is beneficial to increase of growth, and fresh air is of importance. The maximum gain takes place between July and October.

**Growth in Length.**—The length of the newborn child varies according to its weight, degree of development, and the duration of gestation. The measurement in inches is equivalent to the lunar month of gestation

multiplied by two. This gives an average of 20 inches at term for a male infant, the female being about  $\frac{1}{2}$  in. less. Subsequent rate of growth is about  $\frac{3}{4}$  in. per month during the first year. The length is doubled in five years. Growth takes place most rapidly during the early months of the year, from March up till the end of June, and is less rapid in the winter months. The yearly increase is from 2-3 inches for many years. A period of rapid growth in height is often preceded by considerable increase in weight, and accompanied by retardation of gain in weight or even actual loss. The two processes rarely take place at the same time, the body appearing to store up material preparatory to increase in height. Series of figures representing the weight and height of children of different ages are of very little value. Individual differences depend upon racial and family tendencies. The size of the child must be considered in relation to that of the parents, and other children in the family, and more importance attached to the relative progress from month to month, and year to year, rather than to the actual measurements in comparison with other children. As soon as the period of infancy is passed, the rate of growth may be so great that a child appears to be rapidly wasting. Occasionally delay in growth in height may be the only indication of thyroid deficiency.



## CHAPTER II.

### HEREDITARY AND ANTE-NATAL CONDITIONS.

*Heredity—Pre-conceptional Factors—Intra-uterine or Congenital Factors—Mental Impressions—Direct Transmission of Disease—Immunity—Treatment.*

The child is the product of inheritance modified by environment, a house composed of many bricks, a mosaic of many ancestors. Like tends to beget like, an idea expressed in the biblical sayings that “you cannot grow grapes from thorns, nor figs from thistles,” and “the sins of the fathers are visited on the children even unto the third and fourth generations.” The first quotation indicates true heredity, and the other contains the idea of the transmission of acquired characters. It is evident that the scientific study and care of children includes a knowledge of the factors which are liable to affect them before birth, and even before conception. Healthy infants imply healthy parents, and healthy conditions during intra-uterine life. The socialistic statement that all men are equal implies a profound ignorance of heredity, as well as of physiology and pathology. Yet there is an hereditary tendency to health and to a normal average. Sex, family, and racial peculiarities arise through a process of selection, a survival of the fittest, or an inheritance of adaptations and mutations. A racial tendency to particular diseases is sometimes characteristic, e.g., amaurotic family idiocy among the Jews.

In the consideration of abnormal heredity we have to bear in mind the occurrence of variations, fluctuating in degree and kind, and not breeding true; mutations of any size and degree, which are transmitted; and the possibility of the transmission of acquired characters. Many of the lesions transmitted are omissions in evolution, flaws in development, and not acquired defects.

We must, therefore, investigate the pre-conceptional factors, i.e., heredity; the intra-uterine or congenital factors, and their modification by the state of health and environment of the mother during pregnancy; the natal factors, strictly speaking, those which affect the child during birth and described subsequently. After birth the child is exposed to all those factors in the etiology of development and disease which are summed up under the name of environment. It passes from an existence in the dark,



almost entirely protected from violence, surrounded by a fluid medium of constant high temperature, into surroundings of a totally different character. It acquires a separate existence, and becomes an individual with multiple possibilities for good and evil.

Its previous existence may be divided into three stages :—

(1) *The germinal stage*, including :—

- (a) The germ cells, the ovum and spermatozoon, liable to damage by constitutional disease at the time of or before conception ;
- (b) The pre-embryonic period, before the differentiation of the impregnated ovum has begun, during which it is liable to be damaged by morbid agents in the maternal blood.

(2) *The embryonic stage* of 5-7 weeks during which the different organs are formed and may be affected, viâ the placental attachment, after the third week of intra-uterine life.

(3) *The fœtal stage*, that of growth, lasting 32-34 weeks, but not abruptly separated from the embryonic stage.

The inheritance of disease, etc., should be clearly distinguished from transmission. Heredity affects the germinal period. Malformations and monstrosities occur in the embryonic stage, but the liability to them may depend on hereditary defects in the germ plasm. Later, the child is liable to the same diseases as after birth. Many peculiarities are acquired, arise *de novo*, are due to extraneous agencies. For instance, deformities may be caused by amniotic bands.

Various theories of heredity have been advanced. Lamarck, in 1809, held that species are not fixed, but are evolved from earlier and generally less organised forms, and he ascribed the transformation to the effect of use or disuse. According to Darwin, 1850, organisms vary, and no two are alike. They tend to increase to the utmost limits of subsistence, and in the struggle for existence only the fittest survive, a process of "Natural Selection." In 1868 he advanced the theory of "Pangenesis," according to which every cell of the body throws off gemmules, which accumulate in the reproductive organs and grow into similar cells. Neumann, in 1884, put forward the theory of "Cellular Continuity." The germ cells are separated early from the general body cells, remain unchanged, and are transmitted directly from parent to offspring. Weismann, in 1885, argued that heredity is due to "Continuity of the Germ Plasm." He assumed that the reproductive cell consists of two parts ; one, somatic, from which the body cells are formed ; the other, reproductive, remaining inactive and giving rise to the next generation. Thus a father and son are relatively brothers. He maintained that acquired characters are not transmissible. Hereditary modifications may be somatogenic or germinal in origin, due to a potentiality for variation dependent on environment. The germ plasm is situated in the chromosomes of the nucleus, and the number of the chromosomes varies in different animals, but is constant in the same



species. The number in an ovum or sperm cell is half that of a body cell, and union is necessary to raise the number to that of a body cell. He regarded the chromosomes as made up of an immense number of small parts or "determinants," and the characters of the offspring as dependent on the relative combinations and arrangements of these male and female determinants. Hence arise variations.

The most interesting theory of heredity is that advocated by Mendel, and it has the great advantage of being supported by many experimental data. His conclusions are based on the results of experiments on the edible pea, in order to discover the law of inheritance in hybrid varieties. Of two contrasting characters, e.g., size, colour, shape of seed, there is always one in each cross which appears exclusively in the offspring, and this he called the "dominant," and one which does not appear, named by him the "recessive." A dominant parent only produces dominant "gametes," and a recessive produces recessives. Therefore, the offspring are dominants.

Thus.— $D \times R = D (R)$ , the recessive being latent.

On allowing the hybrids to become self-fertilised the characters of both parents appear in the offspring in the form of one pure dominant, one pure recessive, and two impure dominants; that is, apparently dominants but containing a latent and unrecognisable recessive factor. This proves that the recessive character is present, though latent, in the hybrid produced by the union of a dominant and a recessive. From this it follows that two kinds of germ cells are produced by the hybrids, the parental characters being segregated into dominant and recessive "gametes" in equal numbers. The union of a dominant sperm cell (male gamete) with a dominant ovule (female gamete) produces a pure dominant. A pure recessive is due to the union of a recessive with a recessive. The impure dominants are formed by the union of a dominant male or female gamete with a recessive female or male gamete. The pure dominants and recessives are called "homozygotes," and the impure dominants "heterozygotes." The dominant factor is present in three out of four zygotes. The descent may be tabulated thus:—

D (R).					
MALE. GAMETES.			FEMALE.		
D	unites with	D	to form	DD	Homozygote.
D	„ „	R	„ „	DR	Heterozygote.
R	„ „	D	„ „	DR	Heterozygote.
R	„ „	R	„ „	RR	Homozygote.

Each gamete contains only the unsplittable inherited factor, and only one of a contrasting pair, that is, is pure for that factor. The zygotes contain two, being formed by the union of two gametes, and these two characters are splittable, that is, are segregate from each other in the zygote. If similar gametes unite into a zygote, segregation is into like gametes.



But a mixed zygote, a heterozygote or hybrid, contains both factors, and segregates or splits into gametes of each kind. Hence, it is obvious that the mating of hybrids will produce a certain number of pure offspring, which will subsequently breed true.

The blue Andalusian fowl affords an excellent illustration of this principle for it is a hybrid, an impure dominant, and will not breed true.

Blue  $\times$  Blue = 1 Black, 2 Blue, 1 White.

Blue  $\times$  Black = 2 Black, 2 Blue.

Blue  $\times$  White = 2 White, 2 Blue.

Black  $\times$  Black = Black only.

White  $\times$  White = White only.

Black  $\times$  White = Blue only.

Here black is dominant and white recessive. This applies to these colours in the edible pea, and in this plant the quality of tallness is dominant to dwarfness. The descent of the Andalusian fowl supports the statements that pure dominants and recessives breed true; that impure dominants always produce the same kind of offspring as their parents, viz., one dominant, one recessive, two impure dominants; that an impure dominant united with a dominant produces an equal number of pure and impure dominants; that an impure dominant united with a recessive produces an equal number of pure recessives and impure dominants.

These observations have been confirmed in the case of various species of plants, snails, moths, mice, rats, rabbits, and poultry. Immunity and susceptibility to rust in wheat also obey the Mendelian law. The application to the inheritance of the varied characters of man, disease, and deformity, is more difficult, but claims have been made for the accuracy of the theory in the transmission of the colour of the eyes, tufts of white hair, greyness and premature baldness, keratosis or tylosis palmaris et plantaris, epidermolysis bullosa, brachydactyly, the short tail of Manx cats, the aborted coccyx of rumpless fowls, split hand and foot, abortion of the fingers, ptosis, coloboma, cataract, ectopia lentis, night blindness, retinitis pigmentosa, diabetes insipidus, and hereditary chorea. The transmission is generally through an affected parent in whom the peculiarity is a special or dominant factor. Many of the abiotrophies are transmitted, affections which depend on early degeneration before development is completed, such as grey hair and baldness, Friedreich's disease, hereditary optic atrophy, and various myopathies; notably pseudohypertrophic palsy, which is transmitted by females only, and the facial type of Landouzy-Déjérine, which may be transmitted by males. Possibly in the case of pseudohypertrophic palsy the males do not transmit, because they do not reach procreative age. The female transmits it to her sons, but not to all sons. She must, therefore, possess a defect in that part of the ovum which becomes the germ plasm. Similarly, in hæmophilia, the ova of the mother are affected though the body cells are normal.



Possibly other affections will be found to conform to this law, when they have been traced through a sufficient number of generations. Among these may be mentioned asthma and hay fever, exostoses, accessory auricles and helical fistulæ. The law may be disguised or interfered with by the sex factor, as in pseudohypertrophy and hæmophilia; and by the transmitted character being a recessive, perhaps attracting no attention.

**Malformations** in the embryo chick have been produced by shaking the egg, warming one side, varnishing the shell, and the injection of toxins. So, too, interference with the mammalian embryo in the very early stage may cause abnormality, which must be distinguished from an inherited peculiarity. Dimpling may arise from inflammatory adhesions between the amnion and the integuments. Distortions and amputations may possibly be due to a like cause or to constriction by the umbilical cord.

Reference may be made to some of the affections above mentioned. Slight or gross peculiarities of structure are often transmitted through several generations. A family likeness is constantly seen, especially if there is a characteristic feature, such as the nose or chin. Sometimes the resemblance is to a more distant ancestor than the parent. This is spoken of as **Reversion**. **Atavism** is the appearance of a character indicative of the evolutionary ancestry of the species. Atavistic tendencies are present in all infants. The newly born babe, even though premature, is strikingly strong in the hands and arms, and in many cases can support its own weight for as long as two minutes. Its upper limbs are relatively more developed than the lower ones. This has been assigned as evidence of descent from a tree-climbing ancestor, since it would be essential for the infant to be able to cling firmly to its mother when climbing or escaping from her foes. It is not due to the peculiarities of the foetal circulation, for this is the same in mammals in whom the hind limbs predominate in growth. Possibly, congenital hyperplasia of the pylorus, and rumination or merycism, are evidence of a still more remote descent from a vegetarian ancestor. Polymastia and polythelia are probably atavistic.

The transmission of characteristic habits, attitudes, and gaits, may be the result of conscious or unconscious imitation. Often this explanation can be excluded by the death or disappearance of the parent; or the peculiarity may have been transmitted from a remote ancestor. Moral and psychical characters follow the same law. It is a happy explanation when a parent can satisfy himself that unpleasant deviations from the normal in the temper, intellect, or morals of the child, are the result of a reversion to a more distant ancestor. Generally it is the parent from whom the peculiarity is inherited. Often it is due to unsuitable methods of education, bad example, and unsatisfactory environment.

A trivial peculiarity may be transmitted in a most persistent manner. A tuft of white hair has been handed on, always in the same shape, to the first and last children of the family for seven generations. In another



instance it was transmitted by unaffected daughters to their sons. In a third family it was only present in alternate children. In a family of piebalds, studied for six generations, 24 out of 138 individuals exhibited a partial albinism, a white forelock and an underlying white patch of skin down the centre of the forehead. Transmission was by direct descent; in one case to all the children (N. Bishop Harman). In albinism there is a lack of pigment forming power. The descent of this is peculiar. Thus, two apparently normal parents had seven albino children out of eight. In a negro family (Farabee) the union of a normal and an albino negro produced three normal sons of whom two bred true. The third married twice, and had one albino child out of six by the first wife, and three out of nine by the second wife. Both these wives must have carried albinism as a recessive and, indeed, the peculiarity was by no means uncommon in the locality. Another difficulty in the study of albinism is the tendency to regard those in whom pigment is deficient as partial albinos. Darwin noted a curious fact that albino cats with blue eyes are born deaf. Albinism may be associated with various diseases. It is most likely to appear in the children of first cousins, who are heterozygous and contain the recessive factor.

**Colour-Blindness** also illustrates sex limited descent. Females must be pure in colour-blindness in order to exhibit the defect. Males exhibit it, if they receive one dose, and alone are affected, unless an affected male marries a female who carries the colour-blind factor, pure or impure. The affected and the sisters of the affected transmit it to their sons. The female may be a pure colour-blind or an impure colour-blind, that is, not colour-blind but containing the factor as a recessive. The male may be colour-blind though only having one dose of the factor which is dominant for sex. So, too, the colour-blind female must have a colour-blind father, and will have colour-blind sons. The colour-blind male will have sons, half of whom are colour-blind, and daughters, half of whom can transmit the peculiarity.

Mendel's work strongly supports Weismann's views on the transmission of characters by the germ cells (germinal or blastogenic), and not by modification of the body cells (somatogenic). **Acquired characters** are not transmitted, and changes in type can only arise by modification of the germ cell. Superficial and slight mutilations are unlikely to be transmitted, for they do not affect the composition of the blood, and, therefore, cannot affect the germ cells. Centuries of circumcision have left the foreskin of Hebrew children unaltered. Serious damage to nerve tissues and viscera is said to cause the transmission of abnormality in such organs. Massin states that severe damage to the liver of rabbits, bitches, and guinea pigs may result in affections of the liver in the offspring. A form of epilepsy, due to severe injury to the sciatic nerves in guinea pigs, can be transmitted to the offspring, and, according to Brown-Séquard, a similar form of spinal



epilepsy can be produced by hemisection of the dorsal cord in these animals, and is transmissible. There are many instances of new types arising, appearing quite suddenly as "**Sports**," and breeding true to their particular type. The white sweet pea developed suddenly as a sport or mutation from a purple parent, long before any of the intermediate grades in colour. Other similar instances are the Ancon race of sheep, now extinct, the Merino sheep, the blotched tabby cat, and the dwarf bean. In the realm of medicine we can quote deformities, such as webbed fingers, polydactyly, brachydactyly, split foot, cleft palate, microcephaly, Mongolian idiocy, congenital hernia, and diseases, such as alkaptonuria, hæmophilia, and so on. Brachydactyly has been traced through seven generations of 174 individuals (Drinkwater). Half the children of abnormal parents were affected, but the unaffected did not transmit the disease.

From the above considerations we may state that congenital abnormalities, consistent with life and the propagation of the species, may be transmitted and become permanent in the family, provided that they arise as mutations or sports, due to some germinal factor. Mutilations are not reproduced. **Variations** in the offspring are due to the intermixture of dissimilar germ plasma, the spermatozoon and the ovum. They depend greatly on selective breeding, for instance, in pigeons, horses, dogs, cats, sheep, cattle, etc. Pigeons revert to the common rock pigeon type if allowed to breed indiscriminately. Hereditary diseases and malformations can only be prevented by not allowing those affected, or possessing the hereditary factor as a recessive, to have offspring.

#### **Preconceptional conditions affecting the Germ Plasm, Male or Female.**

—It is almost certain that the constitutional state of the parents, before or at the time of conception, may affect the germ plasm. Any change in the blood or tissues of either parent, whether by toxins, ferments, poisons, or general privation, may affect the nutrition of the germ cells, and thus exert an influence on the offspring. Similar influences may affect the fertilised ovum, *viâ* the maternal tissues, in all stages of its existence. Such nutritive vacillations are likely to cause variations and mutations in the offspring.

Consanguineous marriages are not to be countenanced as a general rule, for consanguinity emphasises heredity. Yet the marriages of first cousins should not be prohibited, if the family is certainly sound. There is nothing in such marriages which necessarily tends towards deterioration in the progeny, for sound health is transmitted equally well as ill health. When ill results arise they are due to the effect of a hereditary defect which is naturally present in both parents. Insanity in the parent, if breaking out in the mother during pregnancy, causes an unduly high mortality in the offspring. Apart from this, it has no effect on the mental or physical state of the child *per se*, but if the insanity is dependent on a morbid heredity, this influence may be transmitted to the child, and cause insanity or allied affections in later life. If only one parent is insane, and



there is no family history, that is, if the insanity is due to an accidental factor and is an acquired disease, the child is likely to be unaffected.

Even if one or both parents are insane, and the family tendency is pronounced, it is rare for all the children to be affected. On the other hand mental deficiency in the offspring may depend on disease in the mother, such as cretinism, on alcoholism in one or both parents, or on injury or disease at and after birth. Often no explanation can be found.

In estimating the effect of poisons such as alcohol, lead, opium, and the toxins of disease, we have to remember that the poison may affect the sperm cell or ovum before or at the time of impregnation, and the impregnated ovum during its nine months existence in the maternal womb. Hence the continued action of the poison on the mother is more likely to damage the offspring, through the medium of the blood supply, than paternal poisoning. **Alcohol** has acquired a very bad reputation in this respect. Many sources of error arise in judging its effect. It is even asserted that there is no evidence that parental drinking is a cause of inherited weakness, and there is no inherent reason why germ cells should be affected by alcohol to such an extent as to be incapable of recovery. Many secondary causes come into play after the birth of a child of alcoholic parentage. A drinking parent is liable to neglect the child. Food, clothing, warmth, and general hygiene are likely to be bad or insufficient, and the child suffers from these secondary factors rather than from a direct inherited debility. Again, the association of alcoholism and mental instability does not prove that the mental state is the result of alcohol. It is quite probable that there is a primary mental defect, and that this is exaggerated secondarily by alcohol and imitation. On the other hand, Robinovitch, of Paris, in 1900, ascribed criminality and immorality to alcoholism in the parents; over 90 per cent. in the father only. Of 127 children, 40 died in infancy or prematurely, and only 27 were in good condition. Wigglesworth found parental intemperance in one-sixth of his insane patients. It is probably as frequently an antecedent of sanity. Crothers, in 1902, stated that parental intemperance gives rise to "a drink craving, a neurosis or a mental defect," and obtained a direct alcoholic heredity in 1080 out of 1744 inebriates. On the whole we may conclude that parental intemperance is often due to a neurotic or insane heredity, and that the combination of these two factors, or of alcoholism with tuberculosis or syphilis, exerts great influence on the vitality, the mental and the physical degeneration of the offspring, and is a cause of idiocy, mental and moral imbecility, feeble-mindedness, and a neurotic temperament.

Lead and copper may be conveyed to the foetus. Arsenic passes the placenta with greater difficulty, and mercury not at all, though it accumulates therein (Porak, 1894). Oliver fed pregnant animals on white lead, causing death of the young in utero, and found lead in the foetal liver. Legrand and Winter, in 1889, reported a remarkable case in which both



parents suffered from plumbism. Five children were stillborn. The sixth lived 15 days, had small sclerotic kidneys, and general circumlobular cirrhosis of the liver, and much lead in this organ. Plumbism is a common cause of abortion or feeble infants. The results are the same, but less severe, when the father is affected than if the mother suffers. The poison, therefore, affects both germ cells and foetus. Diachylon is frequently taken to produce abortion, often with fatal results.

The uric acid diathesis, lithæmia, gravel, renal calculus, and gout are prevalent in many members of a family. Before accepting this as a proof of inheritance, the fact that there is a regional distribution for calculus must be considered, and that the same conditions of life and environment may have been co-existent. Gout appears more liable to affect the younger than the elder children of a family, and at an earlier age period. If this be true, it might be urged as an argument in favour of the transmission of acquired characters.

Chlorosis affects many girls in the same family at about the same age. Perhaps there is a congenital imperfect development of the uterus or ovaries. Virchow has ascribed it to hypoplasia, an unduly small heart and narrow blood vessels; the symptoms not arising until an extra tax falls on these structures at puberty. Bunge holds that the anæmia is due to the body storing up iron in the liver and tissues as a supply for the foetus, if impregnation takes place.

In these affections there is no true inheritance of disease, but rather the inheritance of a predisposition dependent on the inherent qualities of the protoplasm of the germ cells. The future may perchance reveal to us that there is a true heredity, a transmission of ferments or lack of ferments which set up or prevent these affections.

**Intra-Uterine or Congenital Factors.**—Some of the features of this period overlap those of the preconceptional stage. Thus, tuberculosis, syphilis, rheumatism, and other infective disorders may affect the nutrition of the germ cell before conception, the impregnated ovum, and the foetus in its intra-uterine life. The better the general environment of the mother, the better is it for the child *in utero*, though healthy babies have been, and will continue to be born under the most deleterious conditions affecting the mother during pregnancy. A liberal diet is important, for the nutrition of the child depends on the blood and lymph conveying nutriment to the ovum and foetus. In tadpoles an alteration in the diet may determine the sex. The effect of alcohol has been discussed above. Exercise is good in moderation, for it maintains the general health and helps to prevent constipation. Hard work should not be continued up to the end of pregnancy. With moderate rest in the later stages, the duration of pregnancy is more likely to be continued up to or beyond full-time, and the infant heavier and better nourished.

All strong emotions and mental excitement should be guarded against.



It is a popular belief that *mental impressions* may be the cause of deformities, although there are no nerve fibres in the umbilical cord to carry the nerve impulses. The idea dates back to the time of Jacob and the flocks of Laban (Genesis, Chap. xxx). **Maternal impressions** are common, but deformities are rare. In many cases there is no history of such an impression. In others the mother is anxious to find some excuse for bearing an imperfect child. It is difficult to admit the possibility of the impression affecting the foetus in the earliest stages of its existence, before the umbilical cord is formed, and yet the different structures are differentiated very early. The cases must be regarded as mere coincidences.

Accidents, injury to the mother, and intra-uterine disease are likely to affect the foetus at all stages of its career, and may cause imperfect development, malformations, intra-uterine death, and miscarriage.

It is still more difficult to believe in Telegony, in spite of the evidence in its favour. According to this theory a female who has been once impregnated by the male may subsequently bear young by another sire, which resemble the sire of the first-born. Such resemblance is more probably due to reversion to a more remote ancestor.

The actual **transmission of infective diseases** is almost, if not quite, impossible, provided that the placenta is healthy. There is not always evidence of disease in this organ, yet it may be sufficiently affected to render it imperfect as a filter. White infarction of the placenta has been ascribed to rheumatism and to salicylates, and is a cause of abortion. In the case of infective organisms, they must either reach the foetus viâ the umbilical vein, or be swallowed with liquor amnii. Infection viâ the vein causes a general blood infection, so the disease in the foetus will have a different distribution to that in the parent. For instance, erysipelas may give rise to endocarditis in the child. Hence, placental infection may mean the transmission of organisms, or of toxins in both directions. The placenta has undoubtedly a selective action, transmitting some poisons more readily than others, and forms a generally efficient barrier.

Ante-natal tuberculosis is illustrative of many points in the action of parental disease on the offspring. Its toxic effects on the germinal plasm may prove a cause of neuroses. The children of tuberculosis parents are often precocious, and abnormally excitable. Morselli, in 1904, ascribed to parental tuberculosis, idiocy or imbecility dependent on cerebral arrest of development, and many other mental defects. Tubercle bacilli sometimes exist in the semen, but it is incredible that a sperm cell could convey the organism and infect the ovum. Much more probably it would impair its nutrition, and render it useless for impregnation. The same argument holds good for the ovum. Possibly teratological results arise from toxic action during the embryonic stage. Many instances of foetal infection are now on record, but congenital tuberculosis must be regarded as distinctly rare. Newborn calves rarely react to tuberculin, and the non-reacting calves



of tuberculous dams do not become tuberculous if they are segregated and properly fed. Schmorl found tuberculous nodules in the placenta in 9 out of 20 tuberculous women, some in the stage of incipient phthisis. This observation suggests that the liability to infection is greater than generally supposed. Sitzenfrey (1909) found tuberculous changes in the vessels of the cord. It must, therefore, be accepted that any tuberculous mother is liable to disease or malnutrition of the placenta, which may then permit the transmission of the bacillus and general blood infection of the foetus. The extent of the disease in the child is proportionate to its intensity in the placenta. It may be limited to a local tumour. The possibility of infection by swallowing liquor amnii must be included. The lungs may escape infection, because they are unexpanded, and have a small blood supply. In late infections, ending in early death of the child, inoculation experiments may be necessary to demonstrate tubercle bacilli in the blood and tissues.

Similar objections to the transmission of syphilis by an ovum or sperm cell, infected by the *Treponema pallidum*, hold good as in the case of tuberculosis. The sperm cell is devitalised by the disease; perhaps sufficiently to destroy its fertilising power, perhaps enough to render the development of the impregnated ovum unsatisfactory, leading to congenital defects of structure, function, and vitality. The mode of transmission to the offspring is discussed in the chapter on syphilis. It is analogous to that of tuberculosis, almost certainly so since Schaudinn's discovery of the *treponema*.

With the exception of diphtheria pregnant women are less susceptible to specific fevers. The transmission of these fevers, and of other diseases due to ascertained organisms, depends on the duration and severity of the disease, and the effect on the placenta. A general blood infection is produced. In variola and measles the foetus may die before the rash appears, may be born with a rash, may develop a rash after birth though infected *in utero*, or may be infected at birth. Mauriceau, the French obstetrician, was born with the marks of small-pox. Typhoid fever causes abortion in two-thirds of the cases. The foetus may die from acute blood poisoning, and the placenta appear healthy, though containing typhoid bacilli. Ulceration of the agminated glands is exceptional. Measles is likely to cause abortion in over half the cases. The fifth month is the most dangerous time in all fevers. It has even been recorded that the mother has transmitted measles and small-pox, without being herself infected. Such cases are open to suspicion of erroneous diagnosis. The *streptococcus pyogenes* and the bacillus of symptomatic carbuncle have been shown experimentally to pass to the foetus. Hydrophobia may be transmitted, even to only one of a litter of pups (Peroncita and Carita). Erysipelas, glanders, anthrax, cerebro-spinal meningitis, relapsing fever, rheumatic fever, yellow fever, and malaria have all been handed on to the foetus.



A woman had malaria in early pregnancy, and gave birth to a child 4 months after coming to England. At the age of 7 weeks the child was intensely anæmic, and had numerous malarial parasites in the blood (Moffat).

The parasites have been found in the blood of newborn infants of women with malaria. Some develop the fever directly after birth, others later. Some exhibit no symptoms and grow normally. The spleen may be enlarged at birth. The later the mother is infected the more likely is the infant to suffer. Paroxysms of shaking during intra-uterine life have been reported by Felkin in two cases, the children being born with enlarged spleens, and having attacks of fever.

**Immunity.**—This is a property due to the formation of antibodies (antitoxin) in the blood and lymph. It is an increased cellular resistance acquired by the leucocytes, not by the germ cells. The antitoxin combines with the specific toxin and arrests its action. The two bodies are not physiological antagonists, though they directly affect and combine with each other; as a rule, but not invariably, in proportionate amounts. The degree of neutralisation may vary in accordance with the degree of concentration or with the duration of contact.

The duration of immunity varies in different diseases and in different individuals. It is often very short in influenza, and may last a lifetime in variola. Two-thirds of the infants of women vaccinated during pregnancy take just as well as usual, and the rest will take in six months.

Congenital immunity is probably always transmitted by the mother, *viâ* the placenta. It is improbable that an immune male can transmit immunity to his offspring by a normal female. Experiments have shown that immunity to diphtheria toxins can be transmitted to young guinea pigs from the mother. Ehrlich rendered young mice immune to tetanus, through the mother's milk, by inoculating the mother with serum from an immune horse. Thus, there is a conveyance of antibodies from the maternal blood to the child, a transmission and not an inheritance, not a cellular immunity.

The blood of the child of a woman with typhoid fever may give Widal's reaction, either because the foetus produces its own agglutins or they are transmitted. Possibly the children of tuberculous parentage inherit a slight immunity. The liability to infection is great, but the incidence is very little larger in these children. Limit the chance of infection, and the disease is limited. The age of onset is earlier, unless contact can be excluded. If it is, the liability is no greater than in those of non-tuberculous parents. The acquisition of partial immunity as the result of infected glands, and the survival of the fittest complicate the study of this question. Measles and syphilis become milder in type in the course of years, and are extremely virulent when introduced among a new people. Negroes and Mongols are relatively immune to yellow fever. The English are more susceptible than the Boers to typhoid fever. Thus, it is probably mainly due to the survival



of the insusceptible. Nevertheless, it is certain that antibodies are transmitted from the mother to the offspring by the maternal blood and by the milk. The liability to infections is much greater in the infants who are not breast-fed, especially during the first few days of life. Immunity from infections in the first weeks of life is probably due to transferred immunity from the mother against the infections she has suffered.

**Treatment.**—Ante-natal treatment is often ignored. The mother must be put under the best available conditions of environment, diet, hygiene, and freedom from mental disturbance. Local and general health must be attended to. Tuberculosis may improve during pregnancy, remain fairly stationary, or progress rapidly and end fatally before confinement. The treatment of syphilis in either parent is essential, and produces excellent results on the offspring. Drugs and other means for procuring abortion must be avoided. They generally fail, produce serious or fatal illness in the mother, or result in the birth of a defective or malformed infant.

## CHAPTER III.

### DIET AND NUTRITION.

*The Mother or Wet Nurse—Physiology and Chemistry—Breast Feeding—Human Milk—Cow's Milk—Bacteriology—The Effect of Heat and Attenuants—Subsidiary Methods of Feeding—Proprietary Foods—Diet after Weaning; in Early Childhood; in School Life; in Illness.*

**The Mother of the Child, or Wet Nurse.**—Every child should be brought up on human milk, preferably by the mother, and especially during the first three months of life. For a few days after birth the milk, known as the *Colostrum*, appears to contain antigens, substances which produce antibodies in the blood of the child, rendering it much less liable to infection. Maternal nursing is particularly important during this period, for these antigens disappear almost entirely in the course of a week or two. The mortality of infants depends mainly on digestive disturbances, which result from an unsuitable diet, and are much less apt to occur in the breast-fed. Maternal nursing conduces to the complete involution of the uterus, and sometimes lessens the chance of impregnation at too short intervals. It is also beneficial to the mother through the mode of life and regular diet necessary, and gives her a much greater sense of responsibility in the management of the child. Even if the mother cannot suckle the child entirely, or for a prolonged period, it is almost invariably advantageous that she should do so to the best of her ability, and should not neglect this important duty for what are often trivial or inexcusable causes. Economic conditions may prevent the mother nursing, because she is compelled to go out to work.

A wet nurse is often the only means of saving the child's life, but is by no means always reliable or available. Such a nurse is an expensive luxury, and may have to be obtained from Paris. Her milk will not necessarily suit the baby for whom it is required. There is no fear that immoral or inborn propensities can be transmitted by the milk to the child.

Seeing that the care of the child begins before birth, and even before conception, the mode of life of the expectant mother must be supervised. The more healthy the mother, the sounder and more healthy will be the offspring. The diet should be rather more generous than at other times, and contain a liberal supply of lime salts, protein, and foods suitable to maintain regular action of the bowels. A little more milk, meat, and bread



will supply all the nutriment necessary for the growing foetus. Alcohol is unnecessary, and, taken in excess, may be injurious. Occasionally, mild purgatives, such as cascara, aloes, liquorice powder, or Apenta water, are required. Violent exercise, lifting heavy weights, any mode of progression causing much jolting, and sea-sickness must be avoided. Regular walking exercise at a moderate pace should be taken morning and afternoon, and continued up to the end of pregnancy, unless rendered impossible by swelling of the legs or pain. Swimming is permissible. The relief from household worries and troubles, and the advantages derived from rest and good food by women admitted to Lying-in Hospitals for at least 10 days before confinement, are shown in the increased average weight of their children.

Mental excitement and emotion, if excessive, are deleterious, but most ordinary amusements and entertainments are valuable in preventing the mother thinking too much about her condition. All the usual hygienic factors of ordinary life are more necessary for the expectant mother. She should rest for at least an hour in the middle of the day during the later months of pregnancy, and have a good long rest at night. Her clothing must not interfere with the respiratory movements or exert pressure or friction on the breasts and nipples. A broad strong jean or flannel bandage may be worn to support the lower part of the abdomen.

The breasts and nipples must be kept clean, and, if pendulous, the folds below the breasts must be freely dusted night and morning with Emol Keleet, zinc oxide and starch, or other drying powder. For three months before confinement the nipples should be washed daily in warm water, and massaged with pure vaseline, lanoline, or cacao-butter. No astringent or spirituous lotion should be used, for it hardens the skin, and makes it much more liable to crack. Small and retracted nipples can be increased in size and drawn out by careful manipulation, and rendered more prominent by the gentle use of Bier's suction cups. During the later months swollen prominent nipples must be protected by absorbent salicylic wool, frequently changed if there is much overflow of milk.

Most drugs can be taken in moderation with impunity, but strong purgatives, large doses of quinine, ergot of rye, and similar drugs, must be avoided. Salicylates are harmless, although they have been supposed to give rise to the "white infarction" of the placenta, which is liable to occur in rheumatic women, causing intra-uterine death of the child.

The perfect mother or wet nurse is 25 to 35 years of age, truthful, placid, and equable in temper, cheerful, good-natured, affectionate, unemotional or well controlled, and active. She should already have had one healthy child, and experience in the mode of management. Many previous pregnancies, in rapid succession, impair the general health and quality of the milk. She should be temperate in food and drink, though not necessarily a total abstainer. Above all she must have plenty of patience, and realise



that for the time being nursing is her paramount duty, and that all other duties and amusements must be put on one side if they interfere with the proper performance of her functions as a nurse. She should present on examination the appearances of perfect health; be strong, robust, and well nourished. There is no advantage in being fat, and, indeed, some thin women are most excellent nurses. The teeth should be good, free from caries, appetite and digestion excellent, and bowels open daily without the aid of medicine. She should be in the habit of taking daily exercise and sleeping well. In the case of a wet nurse more particularly, there should be no evidence of past or present constitutional disease, more especially tuberculosis, congenital or acquired syphilis, gonorrhœa, and former rickets. The mouth and throat must be carefully examined; and the hair and skin, to see that she is free from rashes and vermin. The physical examination must never be omitted, but it is an absurd refinement to apply the tuberculin test, as has been suggested. The nurse's child, if living, should be examined for evidence of disease, and as regards its general nutrition and cleanliness. For this reason it should be at least a month or six weeks of age. The child should be under six months, but it is not necessary that it should be of the same age as the infant for whom the nurse is required. Beware of a substituted child. Chemical and microscopical examination of the milk are unnecessary.

The breasts of a primipara are generally conical or pyriform in shape, not necessarily large, firm to the touch, with prominent nipples. In a multipara, and sometimes in a primipara, they are pendulous. The actual size of the breast is no measure of its functional capacity, for the glandular structure may be relatively small in proportion to the amount of fat. A large breast of this kind diminishes little in size during suckling, whereas a well-developed gland becomes smaller and less tense. Even this is not an absolutely reliable indication of the functional value, for in some women the secretion of milk takes place rapidly during the process of suckling, and only to a slight extent during the intervening period. By weighing the mother's child before and after nursing, on several occasions, a fair estimate of the secretory activity of the breast can be arrived at. The nipples should be prominent and erectile. Occasionally they are too large, but more commonly they are too small, or flat and retracted, rendering suckling a mechanical impossibility.

While nursing, the mode of life should be regular and placid, and include personal cleanliness as well as a certain amount of exercise or physical work daily. The diet should be generous, nutritious, and easily assimilable, but excess of food, especially indigestible food, strongly aromatic foods, highly seasoned dishes, and strong tea, are to be avoided. Wet nurses constantly are overfed. Having previously been in reduced circumstances, and on a plain and insufficient diet, as soon as they get the



chance they drink too much milk, eat an excess of meat, and often take an undue supply of stout or other malt liquor, on the plea that it is essential in order to provide good milk for the baby. The usual result is that the quality of the milk becomes such that it upsets the digestion of the child. As a general principle alcohol is unnecessary, but one or two glasses of burgundy or port, or  $\frac{1}{2}$  to 1 pint of light ale or stout, may be taken daily by women who are accustomed to it. An extra pint of milk in the course of the day, and the ordinary three meals are quite sufficient under most conditions. Bread and milk, gruel, or cocoa may be taken at bed time. Fruit and vegetables may be given freely to counteract the tendency to constipation, common when an excess of milk is taken. Sixty grammes of extra protein in the diet will supply the 20 grammes in a litre of human milk, and a surplus of carbon available for the formation of fat. During the early days of lactation, while the mother is in bed, the diet should be light, easy of digestion, and not too nutritious. Many mothers take too much milk and an excess of food, which, combined with lack of exercise, leads to the secretion of milk rich in solids, and sets up indigestion in the child.

If the wet nurse's child is alive, the employer should see that it is properly cared for, and the nurse consequently free from anxiety. A moral obligation lies upon the employer not to allow the child to suffer while he is paying the mother to deny it its natural food supply. The mortality among these children is enormous, most of them dying under three months of age. For this reason a wet nurse should be preferred whose child has reached the fourth month in life. If the supply of milk is overabundant, it must be drawn off, or given to another child, otherwise stasis and loss of function result. This is especially important when the foster child is a weak or newborn baby, for it is not at all likely to be able to empty the breasts. Under such circumstances it is advisable to allow the nurse to suckle her own child, either at the beginning of or at the end of the nursing period, according to which portion of the milk is required for the foster child. The nurse must not be allowed to go home for this purpose, for she might convey infectious disease, and all control is lost over her diet and habits of life. She must be watched to see that she does not attempt to supplement any deficiency in her milk supply by giving cow's milk or other food, and that she does not soothe an infant irritable from indigestion or insufficient food by means of opiates. The child must be watched to see that it is not overfed.

It is a pure experiment in every case where a wet nurse is tried for a delicate infant. If the child is extremely weak it may be too dangerous to attempt it, and safer to rely upon carefully supervised substitute feeding.

**Physiology and Physiological Chemistry.**—Sometimes a few drops of milk can be squeezed out of the gland during the early months of pregnancy. Usually secretion is active in the last few months, and on pressure milk



will flow out in jets from the nipple. That secreted during the first three days after delivery (*Colostrum*) is scanty, nutritive, and laxative. Active secretion generally begins on the third or fourth day, and increases with the age of the child. It is most active while the child is suckling. Females, human and animal, occasionally secrete milk without having been previously pregnant. In rare instances the male has done so.

The secretion of milk is not a process of filtration. It is the result of the metabolic activity of the secretory cells of the mammary gland. These cells manufacture the milk from the nutrient materials brought to them by the blood. That the fat of milk is not derived from the fat in the food is evident in many cases. Cows out at grass yield much more butter fat than can be accounted for by the fat in the food, and carnivorous animals secrete a milk rich in fat. Generally speaking, the quantity of fat in milk is increased by protein and diminished by fat in the diet. Protein increases metabolic activity, whereas fatty food diminishes it. Possibly, some of the fat may be taken up by the gland cells from the blood directly.

Caseinogen, another constituent of milk, is not found in the blood, and must, therefore, be formed by the metabolism of the cells. The lact-albumin is closely allied to the serum-albumin of the blood, but differs in several respects. It is probably formed or modified by the cellular metabolism. If the normal activity of the gland is disturbed, the percentage of albumin is increased at the expense of the caseinogen.

The lactose or milk sugar is not found in any other part of the body, except in the alimentary canal when it has been taken as food, nor has it been found in the blood. In carnivora it is present in abundance in the milk, although little carbohydrate food is taken. In these animals, it is clear that the lactose is little, if at all, dependent upon the consumption of carbohydrates.

All these considerations prove that milk is a true secretion dependent upon cell activity, that the metabolic processes in the cells are influenced by the nature of the food, and that an increase of one particular food stuff does not necessarily produce an increase of the same constituent in the milk. Like other secretions it is controlled and modified by the nervous system.

Proportionately to its weight the child requires more food than the adult, in order to provide for growth and development as well as for energy in the form of work and heat, the supply of secretions, the repair of waste, and the general maintenance of nutrition. More heat-producing food is required during the early months of life to counterbalance the deficient heat-production from lack of muscular energy, and the great loss of heat by the skin, relatively to the bulk of the body. The loss of heat by the skin varies directly as the superficial area, and inversely as the body weight; the percentage loss is consequently greater in small animals than in larger



ones. In proportion to weight the superficial area in the baby is quite three times greater than in the adult. More than two-thirds of the loss of heat is due to conduction, radiation and evaporation from the surface.

The value of a food is equal to the sum of the values of its component parts, or *proximate principles*, namely, water, proteins, carbohydrates, fats, and salts. It also depends on the nature of the food, assimilability, mode of preparation and administration, the composition of the digestive juices, the condition of the absorbing surface, and the age and idiosyncrasy of the individual.

*Water* is lost in sweat, the expired air, and the excreta. The infant requires a liberal supply because of the extent of the body surface. When the ordinary processes, by which water is eliminated, are increased, more fluid should be supplied, e.g., the sweating of rickets and febrile conditions, in respiratory affections, polyuria, and infantile diarrhoea. Infants frequently suffer from thirst, and should be given plain boiled water, hot or cold, not iced, rather than milk for its relief. Water assists digestion, increases the various secretions, is of value as a solvent and diluent of food substances, and assists in absorption from the alimentary canal. It aids in elimination, keeps the blood in a fluid condition and lessens the liability to thrombosis, and promotes activity of the circulation of fluids and of cell metabolism.

*Protein* food is the source of nitrogen, and is essential to the structure of protoplasm and every body cell. A growing child requires a free supply, relatively more than the adult. Development of young animals depend very much on the percentage of protein in the milk. An infant doubles its weight in 5 months on a 1-2 per cent. protein diet, a calf doubles its weight in 1 or 2 months on 4 per cent. Possibly the albumin is merely nutritive, and the casein is essential for growth. An adult man weighing 67 kilogrammes requires about 100 grammes of protein daily. A six months old baby, one-tenth of the weight, takes a litre of milk containing 20 grammes of protein. For the growing animal vegetable proteins, though free from crystalline extractives, are not as valuable nor as digestible as animal proteins.

An excess of protein in the milk is liable to set up indigestion, colic, and constipation, or simply loss in weight. The stools are green or yellow, and contain curds, especially if the caseinogen is in excess. Excess of uric acid in the urine, gravel, and renal colic may occur. Evil results are uncommon in older children because of their active metabolism. Headache, migraine, and renal colic may be due to this cause.

On a diet defective in protein the child becomes anæmic, languid, weak, and short of breath on exertion. The muscles are flabby and soft, dentition is delayed, the child ceases to grow, and rickets may develop. The absence of the ordinary gain in weight and height is a fair indication that the diet



of school children is deficient in protein. In breast-fed infants, and those fed on whey, a low percentage of protein may lead to curds in the stools, disappearing when more protein is added. This has been ascribed to the formation of an insoluble curd by uncombined acid, or to the abnormality in the relative proportions of fat and protein. Caseinogen with a dilute acid forms a loose precipitate completely soluble in sodium chloride solution, but with an excess of acid an insoluble precipitate is formed. With a low percentage of protein in the food there is liable to be an excess of free acid.

*Fat* is present in all tissues, especially adipose and nervous tissues and bone marrow. Fat in the food is chiefly of value for the maintenance of the body temperature. There is no proof that the fat in the tissues is stored up from the fat taken as food, but the stored-up fat is soon drawn upon if the diet is deficient in this respect, and hence the tissues may suffer indirectly. Fat aids in the absorption of inorganic salts, especially earthy phosphates, from the alimentary canal, more of these salts being found in the fæces if the fat is deficient in the food. It delays protein digestion, lessens gastric peristalsis, diminishes the secretion of gastric juice, and is partially split up by gastric juice and the enzyme *lipase*. In the duodenum it is split up into fatty acids and glycerine. As a rule it is very completely absorbed, but there is often more fat in human milk and other diets than is necessary, from 4 to 5 per cent. escaping digestion, and appearing in the stools in the form of fatty acids chiefly, neutral fats and soaps.

An excess of fat interferes with the gastric digestion of protein, and may lead to "fat dyspepsia" or "fat diarrhœa." The dyspepsia is characterised by constant vomiting of curdled milk and mucus, with the odour of rancid cream, due to butyric acid. Vomiting occurs from half-an-hour to an hour after food, and the fat may resemble lumps of casein. If there is diarrhœa, the stools are loose, yellowish green, acid, greasy, and contain much mucus. It is usually associated with colic, flatulence, intestinal catarrh, and loss of weight. Other symptoms ascribed to excess of fat are refusal of food, capricious appetite, constipation with pasty, foul stools, excess of ammonia in the urine, irritability, and convulsions. These children are often excessively fat, and have a much enlarged liver.

Deficiency of fat impairs digestion and nutrition, causes constipation, and the appearance of earthy phosphates in the stools. It is generally regarded as the cause of rickets, possibly by interfering with the absorption of lime salts in the form of lime soaps. The deficiency in fat cannot be replaced by additional carbohydrates with success.

*Carbohydrates* are of use for the production of heat and muscular energy. Infants practically never suffer from deficiency of this food, for the percentage in milk varies within comparatively small limits. An excess of sugar in human milk rarely produces ill effects, though the infant may become very fat. Artificially fed babies, especially those fed on condensed



milk, often become fat, flabby, unwieldy, and rachitic. They suffer from intestinal disturbances, and fermentation of the food in the alimentary tract, with the production of flatulence and offensive diarrhœa. In artificial feeding either milk sugar or cane sugar is added. Commercial milk sugar is often impure, and liable to be contaminated during preparation. It is probably not identical with that of human milk. On fermentation it is converted into lactic acid. Perhaps there are several kinds of lactic acid, just as there are many varieties of bacilli causing lactic acid fermentation. Cane sugar is cheaper, more handy, and sweeter. On account of its sweetness it cannot be given in such large quantities as lactose. In the small intestine it is converted into dextrose, in which form it is absorbed. Possibly lactose can be absorbed without such conversion, but this is uncertain. There is no serious objection to the use of cane sugar in preference to milk sugar, if it is given in proper amount. The digestive disturbances induced by its use are due to excess rather than to its chemical composition. The starchy foods are referred to later.

*Salts.*—Bunge has shown that the percentages of the salts in the ash of the newly born animal are, with certain exceptions, practically the same as the percentages in the ash of the mother's milk. Milk contains more potassium and less sodium salts, for as the animal grows there is a relative increase of the muscles rich in potassium, and a diminution of the cartilages rich in sodium. The percentage of iron is very much less in the milk than in the newborn. To compensate for this, a young animal stores up iron in the liver previous to its birth, this iron being used up as the animal grows. Animals can live on milk, but die if the salts are extracted. Even if the salts after extraction are again added to the mixture, the animal cannot live on it, for it is unable to utilise the salts except in organic combination with protein. Hence, some of the defects ascribed to deficiency of protein may depend on the deficiency of assimilable salts. When cow's milk is diluted the percentage of iron is reduced below that present in human milk, and may lead to anæmia and debility. To counterbalance this, organic combinations of iron may be given, such as the yolk of egg, raw meat juice, and various proprietary foods made from blood. The iron in the yolk of egg has been estimated as 0·04 per cent. of the dried solids, and as 18·3 mg. per 100 gms. of dried substance. The iron in hæmoglobin is more firmly combined than that in the nucleo-albuminous compound present in yolk of egg. Nevertheless, meat juice and the red gravy from undercooked meat are valuable additions to the diet of anæmic babies. After the age of one year potatoes and small quantities of green vegetable can be added to the diet. Cabbage and spinach contain the highest percentage of iron, and this may partly account for the maxim that "green vegetables are excellent for the blood."



## IRON IN FOODS.

*Milligrammes per 100 grammes of dried Substances.*

White Bread .. ..	1·4	Potatoes .. ..	6·2
Apples, Sweet.. ..	1·7	Green Peas .. ..	6·8
Pears .. ..	2·2	French Beans .. ..	8·5
Cow's Milk .. ..	2·3	Carrots .. ..	8·9
Goat's Milk .. ..	2·5	Lentils .. ..	9·3
Brown Bread .. ..	2·5	Asparagus .. ..	10·5
Red Currants .. ..	3·6	Yolk of Egg .. ..	18·3
Rice .. ..	4·5	Green Chicory .. ..	22·0
Barley .. ..	4·7	Cabbage .. ..	30·5
Black Grapes .. ..	5·8	Spinach .. ..	40·0

—(*The Hospital*, May 16th, 1908).

There is more sodium chloride in cow's milk than in human milk, so common salt need not be added to milk mixtures in artificial feeding. It has, however, some advantages for it delays rennet curdling and makes it less complete. It stimulates the appetite, increases the secretion of hydrochloric acid, and thus assists digestion. Later on, when much vegetable food is taken, the sodium is required to neutralise the potassium in the vegetables. For this reason it is advantageous to add it to barley water. There is one cereal, namely rice, which contains remarkably little potassium.

The percentage of lime in human milk is 0·0243, in cow's milk, 1·51, and in yolk of egg, 0·38. Meats, cereals, and leguminosæ contain very much less. Phosphates of lime and magnesium are most important for cell growth and bone formation. The addition of lime water to milk exerts some influence by virtue of its alkalinity. Lime water does not contain as much lime as cow's milk, and probably lime cannot be absorbed except in the form of organic compounds. Prolonged heat injures milk by rendering the lime salts insoluble.

Phosphorus is of importance in the formation of bones, and, perhaps, in the prevention of rickets. Cereals, leguminosæ, and potatoes contain considerably more than human milk; while lean beef, yolk of egg, and cow's milk contain 6 times as much. Lecithin and nuclein contain phosphorus, and are found in considerable quantities in ova and nervous tissues. It is uncertain whether they are digested and absorbed, but it is harmless to give calves' brain and the hard roes of fishes to children. Nuclein is present in cow's milk, but Koplik states that it is not assimilated by the infant. If more phosphorus is required in the diet it is best given in the form of the yolk of egg. Sulphur is found in cereals, leguminosæ, potatoes, and some fruits, such as cherries and peaches; lettuce and leeks contain a high proportion. Its importance is not thoroughly understood.



*The Chemical Composition of an Infant's Diet.*—A baby, six months old, taking 1,000 grammes of human milk daily, ingests protein 20 gms., fat 40 gms., carbohydrates 66 gms. An adult, ten times as heavy, takes, on an average, protein 100 gms., fat 100 gms., carbohydrates 250 gms. Thus, weight for weight, the infant requires a much more liberal supply of each constituent than the adult does. Halliburton estimates the needs of an infant under a year and a half old at protein 20-36 gms., fat 30-45 gms., and carbohydrates 60-90 gms. The requirements of the individual child are very variable. In regard to protein Waller has pointed out that in proportion to weight the infant requires more than the adult, but in proportion to body surface the amount is approximately the same. Body surface is, therefore, a better proportional indicator than body weight. Weight is a better indicator than age. All three factors should be taken into consideration.

Attempts have been made to regulate the diet according to the calorie value. A calorie is the amount of heat needed to raise 1 kilo. of water 1 degree of centigrade. The number of calories required per kilo. of weight is called the *Energy-Quotient*. Now the calorie value of 1 gramme of protein or sugar is 4.1, and of fat is 9.3. The calorie value of 1 oz. of human milk is about 19, and of cow's milk about 20. One hundred calories per kilo. is equal to 45 per lb. From these figures the necessary calculations can be made. An adult needs from 30-35 calories per kilo. per day. An infant requires during the first 3 months of life 100 calories, during the second 3 months 90-100, and from 6-12 months 80-90 per kilo. daily. Atrophic and premature children may need 120 calories or more. These calculations are interesting, but not of very much value in practice.

**Breast Feeding.**—The child should be put to the breast as soon as the mother has recovered somewhat from the fatigue of labour, say, in 6-12 hours. The excitation of the nipple reflexly induces uterine contraction, and lessens the risk of post-partum hæmorrhage. The child has an opportunity of drawing out the nipples if they are small or retracted, before the rapid swelling of the breast, usually occurring on the third day, makes such nipples so depressed below the surface that it is difficult or impossible for the child to get hold of them. A small amount of colostrum is obtained, valuable for nutrition and its laxative action. It is unnecessary, and often injurious, to give castor oil or other purgatives, or the concoction of butter and brown sugar administered for this purpose. Such treatment may start troublesome gastric and enteric disturbance, which may end fatally. Suckling stimulates the secretion of milk. The amount obtained varies with the vigour of sucking, and the state of the breasts. Exhausted by its entrance into the world, change of temperature and bathing, the newborn sleeps most of the first 24 hours. Usually from one-third to two-thirds of an ounce is taken in two meals in the first 24 hours; 3 oz. in 4-6 meals on the second day; and then 7, 10, 12, 14, and 16 oz. on successive days.



Nothing, not even milk and water is to be given. From the fact that lactation is not established until the third day, and often later, it is evident that no food other than the colostrum is required before that time. Food given diminishes the activity of suckling and the consequent stimulation of milk production. Boiled, cooled water may be given during the first three days, sweetened with saccharin, if necessary to render the urine less concentrated and irritating. If at the end of the third day the supply of milk is insufficient, it may be supplemented by a mixture of milk, cream, water, and milk sugar. The child should be fed every 2 hours, on the breast and the milk mixture alternately, or put to the breast every time and allowed to take some of the mixture afterwards if not satisfied. Weakly and premature infants may have to be fed during the first three days. Begin with a teaspoonful or two of a 5 per cent. solution of milk sugar, and add small quantities of milk and cream gradually, if the child has to be brought up artificially.

During suckling the child should be held partially on its side with the head and back supported on the right or left arm of the mother, according as it is fed from the right or left breast, and the mother must bend her body somewhat forward so that the nipple falls easily into the infant's mouth. The breast must be steadied by the index and middle fingers of the disengaged hand placed above and below the nipple.

Pressure with these fingers will enable the mother to prevent the child taking the milk too quickly, or to assist the flow by gentle pressure on the breast. The child obtains the milk by compression of the sinus and base of the nipple, and not by suction, in a similar manner to the way milk is obtained in milking cows. During the first week the child should be put to both breasts at each nursing, and after that to alternate breasts every other feed, unless it is weakly and the milk supply scanty. The mother should express a little of the milk first to get rid of organisms present in the ducts. The duration of each nursing is from 10-20 minutes, but varies with the strength of the child and its requirements, the rapidity of suckling, the frequency of feeding, the state of the nipple, and the quantity and quality of the milk. A healthy child, taking milk from a full breast, may nurse until satisfied. It should not be allowed to go to sleep with the nipple in its mouth. After nursing, the mouth, especially the corners, should be wiped, but the inside must be left alone for fear of injuring the delicate mucous membrane. During the first few days the infant may be put to the breast every 2 hours, while the mother is awake, or, if very feeble, every  $1\frac{1}{2}$  hours. The stomach is rarely empty in less than 2 hours. It is sometimes better that the breast should be given every 6 hours on the first day, 4 hourly on the second day, and after that every 2 hours. The more frequent nursing during the first two days is advantageous to the child, and stimulates the secretion of milk. When lactation is fully established the child should be given the breast every 2 hours from 5 a.m. to 11 p.m. during the first



month ; every  $2\frac{1}{2}$  hours from 5 a.m. to 10.30 p.m. during the second month ; and after that every 3 hours from 5 a.m. to 11 p.m. Some babies require feeding less frequently, but great circumspection must be employed in lengthening the intervals, and even more in shortening them. It is a common custom to feed the child whenever it cries, on the supposition that the cry indicates hunger. More often the cry is due to indigestion, and the indigestible character of the milk is induced by too frequent nursing. The pain of indigestion can be relieved by any warm fluid, such as hot water, but the warm milk only gives relief by virtue of its warmth and induces fresh colic in a short time. Very feeble infants, and occasionally vigorous ones with active digestive organs, require an extra feed during the six hours interval at night. During the later months of nursing the 5 a.m. feed may be omitted, if the child sleeps soundly until later. Some infants do better on less frequent feeds such as 5 feeds at intervals of 4 hours, sleeping soundly for 8 hours at night. These cases are exceptional, and show the necessity of not rigidly adhering to a hard-and-fast rule, although it applies to the majority of infants.

*Regularity of feeding* is essential to success, for if the intervals are irregular in duration, the quantity and quality of the milk vary considerably. Both the babe and the mammæ should be trained to exact regularity. With a little patience the babe can be accustomed to waking up for its meals with the regularity of clockwork, and sleeping 6 hours at night. Too frequent nursing increases the percentage of proteins in the milk, renders it indigestible, and does not allow the child's stomach sufficient time to digest its contents and pass them on into the duodenum. Wake the child when the feed is due, if it is asleep. If it is difficult to keep it awake, shorten the duration of the nursings, so that in future it will wake up hungry at the proper time. If the interval is too prolonged the child is hungry, sucks greedily, rapidly fills or overfills the stomach, and then vomits or gets an attack of indigestion.

At night the child should sleep in a cot by the side of its nurse, and not in the same room as the mother, if a nurse is available. If the mother is disturbed in the night by the child's cry, the temptation to give the breast is almost irresistible, and likely to be encouraged by a tired husband.

Provided the mother is healthy and strong, and the milk supply satisfactory, suckling may be continued for 9 months, and partial suckling for 10-12 months. Except under special advice it should never be continued after 12 months, and usually it is advisable to discontinue it at the end of 10 months. Few mothers are unable to suckle their children during the important first three months of life, and, if possible, artificial feeding should be postponed until after this period. By the fourth or fifth month the child's stomach is more fully developed as a receptacle for food. Its digestive powers are stronger, and there is less liability to infections.



If the mother's milk becomes unsuitable, it can often be improved by alteration in the diet, the mode of life or the frequency of nursing.

Unduly prolonged suckling is injurious to the mother, and may give rise to headache, anæmia, debility, muscular pains, and increased susceptibility to disease. Amaurosis, epilepsy, and insanity are rare sequels. The child may suffer on account of deterioration in the quality of the milk, a reduction in protein and total solids, and consequent insufficiency of the diet.

Among the lower classes a child is often nursed for 2 or even 3 years on account of the prevalent idea that conception does not occur while the mother is nursing. Various observations show that impregnation does not take place quite so readily during lactation as at other times. From 3·5-8 per cent. of suckling women conceive, although the catamenia have not returned, but the return of the menses is the best indication that the mother may again conceive. Under such circumstances the continuation of suckling has probably little or no effect in preventing impregnation.

Breast feeding may be injurious to either the child or the mother. Gastric disturbance and colic in the infant may be due to irregularity in feeding, milk too rich in protein, or other changes in its composition. Easily digested milk is not always sufficiently nutritious, by reason of its small percentage of solids. Sometimes there are defects in quantity as well as in quality.

The general health of the mother may be unsatisfactory. Tuberculous women do not secrete good milk, but often nurse their children satisfactorily. Nursing must be prohibited if there is active lung mischief. In the presence of latent disease suckling must not be long continued. It is possible that the tubercle bacillus may reach the infant through the milk supply, but is still more likely to be acquired by direct infection, if there is actual lung disease. Past tuberculous disease of glands or bone does not contraindicate nursing, provided the child is weighed every week and the general health of the mother is maintained.

Constitutional syphilis may render the milk supply insufficiently nutritious, but, on account of the delicacy of the baby, the mother must nurse her child and supplement the diet if necessary. A neurotic inheritance is undesirable in that the insanity of lactation may develop, or that melancholia may follow on prolonged lactation. Epilepsy is not necessarily a bar if the mother is under observation, but she might injure her child in a fit, or her milk supply may be affected. The emotional temperament, associated with a neurotic heredity, is liable to cause alterations in the milk on very slight provocation. General debility from any cause may render it necessary to stop nursing. Immediate weaning is essential in acute diseases, especially those of the infective type. The rheumatic pains, sometimes called the "rheumatism of lactation," may be severe and



constant. If they are not cured by a nutritious diet and tonics, the child must be wholly or partly weaned. Albuminuria as a rule does not interfere with nursing. Lactation must not be considered a serious drain on the system, if the food supply is satisfactory and nutrition well maintained. It is important, therefore, never to recommend weaning except after most careful consideration. In doubtful cases nursing should be tried, and the effect on the mother and child carefully noted.

*Local affections of the nipple* may be so severe as to render suckling impossible. Fissure is preceded by abrasion or excoriation due to want of cleanliness, constant moisture from galactorrhœa, too frequent suckling, or the use of hardening astringent lotions. Infection of the raw surface and the mechanical effects of suckling prevent healing, and lead to adenitis. Preventive treatment consists in bathing with cold water before and after nursing, rubbing in olive oil or lanolin, or painting with raw white of egg. Treat excoriation with zinc ointment; or balsam of Peru, one drachm to the ounce of cold cream and lanolin, equal parts; or tannic acid, grs. 15, balsam of Peru, grs. 30, vaseline, 1 oz. For the treatment of fissure dry the nipple carefully and paint it freely with boric acid, grs. 20, to mucilage, 1 oz. If this fails to cure, touch the fissure every other day with a finely pointed stick of nitrate of silver. Or wash with 5 per cent. solution of boric acid, anæsthetise with cocaine, paint on 10 per cent. solution of nitrate of silver, dry with absorbent wool, and paint on egg albumin. For the next few nursings use a glass nipple shield. Sometimes it is necessary because of pain or bleeding to stop nursing entirely for the time being, maintaining the activity of the gland by systematic use of the breast pump. Pain can be relieved by applying a 5 per cent. solution of cocaine half-an-hour before nursing, but the nipple must be washed before the child is fed.

*Lymphangitis of the breast*, abscess, and primary tuberculosis render nursing impossible. In mastitis even the use of the breast pump must be forbidden. In the early stages of congestion of the breast apply hot oil fomentations and massage gently from the periphery towards the nipple. At first it is very painful, but soon milk flows freely, and the child can be put to the breast. If the congestion has proceeded further, a full dose of Epsom salts must be given at once, and two grains doses of quinine 3 or 4 times a day. Equal parts of extract of belladonna and glycerine are applied locally. Surgical interference is necessary if there is evidence of pus. Tuberculosis of the breast may take the form of irregular disseminated nodules, a confluent mass, or miliary tubercles. It sometimes leads to abscess and sinus formation. The axillary glands are enlarged, the affected breast smaller than normal, and the nipple often retracted. It is often painless. Though occasionally seen before puberty it is most common at 20-35 years of age. In a girl of 17 years there was no other evidence of tubercle, and her child was born healthy (E. P. Davis, 1897). About 60



cases are on record. Old mastitis is of importance as an indication that the breast is imperfect and incapable of full physiological activity.

The only reliable evidence of an insufficient milk supply is a lack of gain in weight. Few women anxious to suckle their infants will admit their inability to do so. Occasionally both mother and nurse are deceived on account of the size of the breasts, and the duration of time the baby suckles, although it is wasting rapidly. Weighing the child before and after nursing will give the weight of milk obtained. Chemical and microscopical examinations afford evidence of its quality. Observation of the child while suckling may show where the fault lies. If the infant sucks vigorously for a few minutes and then drops the nipple with an angry cry the quality is probably good but the quantity deficient. If the child nurses languidly and for a long time, the quality is probably poor but the quantity abundant. Disturbed sleep, fretfulness and crying, gastro-intestinal disturbance, and abnormal stools are usually due to milk too rich in protein. This is not an indication for weaning, as it can usually be reduced by regulation of the mother's diet, and decreasing the frequency of nursing, or by giving the child a small quantity of plain boiled water or lime water immediately before it is fed.

**Human Milk.**—Human milk is a thin, watery, bluish-white, sterile fluid, with a peculiar taste and odour. It is generally said to be faintly alkaline, but according to Kerley, Gieschen, and Myers (1903), it is invariably acid if tested with phenol-phthalein. The specific gravity ranges between 1030 and 1035 at 60° F. It varies a little with the temperature. With a low percentage of fat and a high one of sugar it may reach 1042. If the percentage of fat is high it may fall as low as 1024. The specific gravity and the percentage of fat afford a fair estimate of the quality. Milk containing 3-5 per cent. of fat and of average specific gravity may be regarded as good milk.

The fat globules vary in size from 0.00015-0.005 mm. in diameter, and their number is in direct relation to their size; up to 11 millions per cm., averaging 5 millions. The best milk contains a medium number of a medium size, and the worst a small number of small size. It is impossible from microscopical examination alone to say that the milk is of good quality.

Milk varies in composition in different women, on different days, at different periods of the day, at different stages of each nursing, and in the two glands. It is modified by the state of the health, diet, exercise, menstruation, prolonged lactation, and other causes. It contains ferments, such as katalase and antibacterial alexins. In general composition it resembles the mammary secretion of other animals. It does not curdle with rennet, unless a little hydrochloric acid is added, and then only forms a fine flocculent coagulum.

In order to obtain a sample for analysis a breast pump must be



employed, using very gentle suction. It must be thoroughly clean and both nipples and breasts previously washed. The sample used for analysis should be taken from the middle of the nursing; the milk first poured out is watery, and poor in fat, and that obtained at the end is especially rich in fat; hence the conclusions drawn from an isolated partial sample are of comparatively little value. It is very difficult to empty the breast with a breast pump.

*Colostrum* differs from the milk secreted when lactation is fully established. It is yellowish in colour, more alkaline in reaction, of a higher specific gravity, and contains the colostrum corpuscles. These are large nucleated cells, epithelial in character, from the acini of the glands, containing granules and fat globules, but not yet disintegrated. Colostrum also contains mononuclear and polynuclear leucocytes and lymphocytes. The presence of colostrum corpuscles indicates non-establishment or disturbance of equilibrium in the gland. They are variable in number, and may be absent if the mother is feverish. Usually they persist for 7-10 days. They disappear more slowly from the milk of a primipara than a multipara, during bad health and in puerperal affections. They may reappear during ill-health and at the commencement of involution of the gland. If so, the milk will disagree with the infant. Colostrum contains a higher percentage of protein and salts, and a lower percentage of sugar than the later milk secreted. The high percentage of protein is due to the number of corpuscles. The amount of fat is variable, and the fat globules vary in size. Colostrum is coagulable by heat, and may coagulate spontaneously. Its relative composition is unimportant, seeing that it is only secreted for a few days and may vary daily. The importance of the antigens or antibodies which it contains is referred to above (p. 25).

An approximate clinical examination of milk can be made by Holt's method. It is based on the fact that the percentages of salts and sugar are nearly constant, that the percentages of fat and protein vary, and that the specific gravity averages 1031 at 70° F. An increase in the fat lowers the specific gravity, and increase in the protein raises it.

The apparatus consists of a small hydrometer graduated from 1010-1040, a pipette, and a glass-stoppered cylinder graduated in 100 parts and holding about 10 c.c. Half an ounce of milk is required. The specific gravity is taken by the hydrometer. To estimate the fat the glass cylinder is filled by means of the pipette to the upper line exactly. The cylinder is then stoppered and allowed to stand for 24 hours at a temperature 66-72° F. Generally the lower limit of the cream becomes sharply defined in that time, but an additional 6 hours may be allowed if necessary. Chemical examination shows that the ratio of fat to cream is very nearly 3 to 5, and for clinical purposes it may be so estimated. The amount of protein can then be judged from the following table :—



Clinical Examination of Milk (Holt).

	Specific Gravity.	Percentage of Cream.	Protein (Calculated).
Average .. ..	1031	7	1·5 per cent.
Normal variations	1028-1029	8-12	Rich milk.
	1032	5-6	Fair milk.
Abnormal variations	Below 1028	Above 10	About normal.
	„ „	Below 5	Very poor milk.
	Above 1032	Above 10	Very rich milk.
	„ „	Below 5	About normal.

Holt asserts that conclusions drawn from this mode of examination are as exact as those obtained by the ordinary examinations of the urine.

Many analyses of human milk have been recorded, showing marked discrepancies in the percentages of protein and sugar, though comparatively little in the total of these two bodies. In some of the older analyses the percentage of protein is too high, and of sugar too low. The next table shows the results obtained in recent years by analysis of specimens from a large number of women.

Meigs' results are included, because carefully carried out, although he probably underestimated the percentage of protein, and overestimated that of sugar.

The Percentage Composition of Human Milk.

	Meigs.	Sharpless and Darling.	Adriance.	Pfeiffer.	Carter and Droop, Richmond.	Leeds.	Average.
Protein	1·046	1·34	1·48	1·944	1·97	1·995	1·5-2·0
Fat ..	4·283	2·91	3·83	3·107	3·07	4·131	3·0-4·0
Sugar	7·407	7·01	6·72	6·303	6·59	6·936	6·0-7·0
Salts ..	0·101	—	0·17	0·192	0·26	0·201	0·2

Estimations of the relative proportions of caseinogen and lact-albumin are extremely unequal, although in the majority the total of the combined proteins is about the average, e.g.:—



*The Relative Percentages of Caseinogen and Lact-Albumin.*

Observer.	Caseinogen.	Albumin.	Total.
König ..	0·61	1·27	1·88
Hirt .. ..	0·63	1·50	2·13
Lehmann ..	1·20	0·50	1·70
Tolmatscheff	1·28	0·34	1·62
Wynter Blyth	2·40	0·57	2·97

Although milk may vary considerably in its composition it must be regarded as normal if the infant takes it well, digests it satisfactorily, gains weight, and keeps in good health. Harrington found, in the milk of women whose infants were doing well, that the protein varied from 1·08-4·17, fat 2·02-5·16, sugar 5·68-7·30, salts 0·12-0·21. More importance must be attached to the clinical condition of the child than to the chemical analysis of the mother's milk.

Other constituents are found in minute quantities, namely, nuclein, lecithin, cholesterin, neurin, and a yellow lipochrome. Various enzymes are present, but very little is known about them. There are no microbes, except such as have got into the ducts of the nipple from outside, and possibly into the milk in certain infective disorders, such as puerperal sepsis. The salts are present in the proportions suitable to the needs of the growing animal, while the percentage of the sugar is fairly constant, and that of protein and fat more variable. The two latter constituents vary under numerous conditions, and are generally the cause of intestinal derangement or lack of progress in the child.

Human milk may vary in quantity or quality. It may contain an excess of water, or vary in the relative proportions of its different constituents; occasionally it contains deleterious substances. The fore milk, that first secreted, contains more water and much less fat than the strippings, that obtained last from the gland. The strippings of cows is sometimes so rich in fat that it is sold as cream.

The quantity secreted daily varies with the age and needs of the child. At 1-3 months the normal average amount is 600-900 c.c., roughly 1-1½ pints, but this amount is often exceeded. The quantity increases rapidly up to the end of the second month, and then much more slowly. Babies vary in size and appetite and mothers in milk secreting powers. During the first 2 or 3 months large babies take from 15-20 per cent., and small babies 10-15 per cent. of their weight in milk; after that the proportions are smaller. The daily quantity has been calculated from observations by Hæhner, Laure, and Ahlfeld as :—



From the end of the 1st to 4th week	10 to 25 oz.
During the 2nd month .. ..	20 to 30 oz.
From the 3rd to 6th month .. ..	25 to 35 oz.
From the 6th to 9th month .. ..	30 to 40 oz.

The quantity can be increased by stimulation of the breasts, either by use of the breast pump or the suction of other infants ; the ingestion of an increased amount of fluid, preferably nutritious fluid ; the moderate use of stimulants ; extra food, maltine and somatose ; drugs such as iron, arsenic, and strychnia ; faridisation of the breasts ; gentle massage of the breasts for 10 minutes 3 times a day, and massage of the abdomen in an upward direction. Galactagogues are drugs said to directly influence the secretion of milk. Subcutaneous injections of nitrate of pilocarpin, gr.  $\frac{1}{8}$ , for a few successive days may increase diminished secretion or restore one rapidly failing. Drachm doses of tincture of common goat's rue (*galega officinalis*) 5 times a day, and tincture of the stinging nettle in doses of  $\frac{1}{2}$ -1 oz. have been recommended by Grinewitch. Veterinary surgeons give infusion of aniseed freely internally, and apply it to the mammæ as a fomentation. Thyroid extract is sometimes used.

The quantity may be diminished by reducing the amount of liquid in the diet and giving saline cathartics. A full dose will sometimes entirely stop secretion for a time. It is rarely necessary to diminish the quantity of the milk, for if it is too poor in quality it is better to try and improve it by measures which increase the percentages of solids. Preparations of belladonna or atropine taken internally or applied locally lessen the secretion. Camphor, grs. 3-5, 3 times a day for 3 days, may completely arrest the flow, and sometimes potassium iodide lessens it. Complete agalactia may result from fright or sudden shock.

Age has very little effect on the quality. Menstruation may cause an alteration of such a nature as to set up indigestion or diarrhœa in the child. Usually a slight disturbance of the digestion is all that results, but occasionally the infant is seriously affected. More generally the milk is unaltered and the child unaffected. Dyspepsia, colic, and enteric catarrh are more often coincident than due to this cause, and should be treated in the usual way, and not by weaning. If the milk markedly disagrees it will probably be because of an excess of protein, and all that is necessary is to lengthen the interval between the nursings, give a little water to the child before nursing, or temporarily modify the diet and mode of life of the mother.

Should the mother become pregnant while nursing the question of weaning depends on several factors. Supposing the child is gaining weight, and is contented, and the mother's health is not suffering, suckling may be prolonged to the fifth or even the sixth month of pregnancy. If so, the child must be weaned entirely by the end of the month, and, as a rule, at a much earlier date. If the baby is delicate, or the weather hot, suckling should be



continued as long as possible. If the mother is delicate or the milk supply insufficient, partial weaning may be begun at the end of the fourth month. The slight risk of reflex miscarriage being set up by suckling is so small that it may be neglected, except in women who are very prone to miscarry. Few conceptions take place before the sixth month of lactation, and at that age a baby can generally be weaned with little risk, if it is thought advisable for the sake of the foetus, or on account of the health of the mother.

The percentage of fat in milk can be increased by a liberal supply of protein food. Malt extracts also increase the amount of fat. Vegetable diet diminishes both the amount of fat and protein in the milk. An increase of protein in the diet also increases the amount in the milk, and thus makes it more indigestible. To counterbalance this the increased nitrogenous diet must be supplemented by an increase in the amount of exercise taken. To convert a poor milk into a digestible rich one, give a liberal protein diet and walking exercise morning and afternoon, or even order a moderate amount of riding, cycling, and lawn tennis. A nursing mother must not fast. Deficiency of food is bad both for her and the child; the milk is weaker in protein and fat, and the mother has to draw on her reserve stores of nutriment to provide even this imperfect diet.

Alcohol is said to increase the proportion of fat, to slightly diminish the amount of sugar, and to have no constant effect on protein. A moderate amount can be taken without injury and without any alcohol appearing in the milk. It should not be prescribed as a general rule for fear of setting up the alcoholic habit, but in some cases it will help the mother to continue nursing. Good milk depends on suitable food, not upon stimulants. To insist on the prolongation of nursing, when it can only be continued by means of a considerable amount of stimulation, is bad for both mother and child.

Increasing the frequency of nursing increases the percentage of solids, especially protein, in the milk; resulting in colic and indigestion. The more frequent the feeding the more indigestible the milk becomes. By lengthening the intervals between the feeds, the milk may be rendered more digestible.

In order to alter the composition of milk we can adopt the following simple methods. The percentage of protein is increased by increased frequency of nursing, increased protein food, and insufficient exercise; and is decreased by the opposite conditions. The percentage of fat is increased by increased protein food, malt extracts, and possibly alcohol; and is diminished by excess of fatty foods, deficiency of protein and fasting. The percentage of water is increased by an increased fluid diet, and diminished by a lessened ingestion of fluid and the use of saline cathartics.

Certain drugs given by the mouth to the mother are partly excreted in the milk and may affect the child, but the amount of excretion is unreliable and uncertain. Citric acid and hydrochloric acid produce no change in the milk. Sodium salicylate, potassium iodide, iodoform applied



externally, iodine injected intramuscularly, and colchicum may all pass into the milk. Mercury is transmitted only feebly and irregularly. Lead, arsenic, and antimony have been excreted. Rhubarb, senna, and saline cathartics may pass into the milk and purge the child. Opium and morphia taken by the mother have sometimes produced deep sleep in the infant; so, too, chloral hydrate. The effect of bromides is variable. Phenazone, phenacetin, and urotropin may appear in the milk, and, possibly, atropine and belladonna. Attempts to treat the child through the medium of the milk supply, by giving drugs to the mother, are foolish, and uncertain in their results.

**Cow's Milk.**—The general composition of cow's milk is like that of human milk. The percentage composition differs. In addition it is crowded with micro-organisms, and is a very suitable medium for their growth. Its specific gravity ranges between 1028 and 1035. When secreted it has an amphoteric reaction, turning litmus blue and turmeric brown. With phenolphthalein it is acid. With litmus the reaction may be amphoteric, due to the salts, acid and alkaline sodium phosphate. Citrates are more abundant in cow's milk than in human milk, the amount being equivalent to 1·2-2 gms. per litre (Obermeyer). On boiling, the soluble bicitrate of calcium is converted into a less soluble tricitrate. Many ferments are present and are readily destroyed by heat. Microscopical examination reveals extraneous substances, such as epithelial cells, particles of manure hairs, etc.

The total solids vary within considerable limits dependent upon the breed of the cow, the period of lactation, and the nature of the food. The total solids, less fat, vary within comparatively small limits from 8·5-11 per cent. Each cubic millimetre contains in suspension from 2-3 million fat globules. The fat is poorer in olein than is the fat of human milk, but amounts to over 40 per cent. It is a mixture of neutral olein, palmitin, and stearin, and glycerides of the fatty acids, palmitic, stearic, and oleic. Glycerides of certain volatile fatty acids, chiefly butyric, caproic, and caprylic, are present in small quantities. Purin bodies, lecithin, cholesterol, and mineral salts are also present. The sugar is in the form of lactose.

The proteins are caseinogen and albumin. The terminology of proteins varies with different writers. The caseinogen of Halliburton is the same as the casein of German writers, and the free casein of Van Slyke and Hart, i.e., it is equivalent to calcium casein or bicaseinate of calcium, for it is always combined with calcium. The casein of Halliburton is the same as the paracasein of the Germans. Caseinogenate of calcium or basic calcium casein is formed by adding lime water to milk; caseinogenate of K. or Na. by adding alkalies of K. or Na.; basic calcium casein, *plus* antacid, by adding bicarbonate of soda; sodium casein by adding citrate of soda; and calcium lactate in buttermilk. None of these compounds is coagulable by rennet. Calcium casein forms with rennet in a faintly acid medium



calcium paracasein or curd. If the milk is rendered alkaline it will not curdle, but the addition of a small amount of alkali only delays clotting until it is neutralised. Further observations are required on the necessity for and justification of this nomenclature.

The average composition of cow's milk, obtained by different analysts, does not show nearly the same discrepancies as in the case of human milk. Protein is estimated by Kjeldahl's process, fat by Babcock's apparatus or Gerber's acido-butyrometer, and sugar by Fehling's process. Provided the milk has not been tampered with, its specific gravity depends chiefly on the percentage of fat. This varies considerably in different cows, in the same cow at different times, and during the same milking according as the fore milk, middle milk, or strippings is taken for analysis.

The chief fraud in milk dealing consists in the removal of cream. To detect such a fraud place 100 c.c. in a graduated vessel, or fill any glass cylinder graduated in 100 parts, take the specific gravity, and after allowing the milk to stand for 24 hours, read off the proportion of cream which rises to the surface. If the specific gravity is within the limits of 1028 to 1035, and the percentage of cream on standing not less than 9, it may be considered good average milk. The higher the percentage of cream, and the lower the specific gravity, the less likely is the milk to have been tampered with.

The difference between milk and cream is essentially one of fat percentage. There are two kinds of cream : (1) Gravity Cream, obtained by skimming the milk after it has stood for 24 hours ; and (2) Centrifugal Cream, obtained by the use of a separator. The latter is fresher if it is made from fresh milk. Possibly, to a slight extent the emulsion is broken up by the centrifugal force. The richest centrifugal cream contains 40-50 per cent. of fat. Much of it contains 20 per cent. only. Gravity cream is very variable, and usually contains about 16 per cent. fat ; sometimes it is merely rich milk. It does not keep well, and is often preserved by means of ice or the addition of preservatives. If the cream is obtained by skimming, it should be taken from the mixed milk of a herd and not from a fancy cow. The milk should be allowed to stand at the same temperature, for the same length of time, and skimmed by the same person. Centrifugal cream is separated from the mixed milk of many animals. The milk is filtered, cooled to 40° F., pasteurised, centrifuged and again cooled to 40° F. The fat globules may become coherent and indigestible, giving rise to vomiting of water, fat, and curd in succession. This is most liable to occur if the bottles have not been kept cool, or have been much shaken in transportation.

The gravity process can be used for obtaining cream of various percentages of fat. According to Townsend the top quarter of milk, which has stood for 6-8 hours, contains 10 per cent. of fat. The upper half will contain 7-8 per cent. Chapin uses the top 12 oz. of a quart of milk, and says it



contains 12 per cent. of fat. Holt states that the upper half contains 7 per cent. ; the upper third, 10 per cent. ; the upper fourth, 13 per cent. ; and the upper fifth, 15 per cent. of fat, if the milk is allowed to stand 4-5 hours. All cream should be sold according to the percentage of fat it contains. It should be pasteurised at the dairy, cooled down, and kept in ice.

Only the milk from healthy cows, subjected to the tuberculin test, should be used. It is preferable to use mixed milk from a large number of cows, as by this means a more uniform standard quality is obtained. Milk from one cow only is liable to great variation according to the period of lactation, the nature of the food, and the condition of its health. The best cows are Shorthorns, Ayrshires, Kerrys, Devons, and Red Polled. Jersey, Alderney, and Guernsey cows yield a milk containing a higher percentage of fat, but they are more liable to disease. The milk of new calved cows must not be used because the colostrum corpuscles are liable to disagree. The milk of old cows, or of those which are calved for a long time, will be thin, watery, and deficient in fat. Cows should have a liberal supply of clean water, plenty of food, a ration of nitrogenous food, and comparatively little exercise. They must not be fed on fermented food, such as the refuse of breweries, nor allowed to drink stagnant water, and the pastures should be free from noxious weeds. Frequently the milk of cows fed on turnips and linseed or cotton cake disagrees with infants. A diet of hay and oats or of grass is the best. Cow houses should be built on the most approved principles, well drained, kept scrupulously clean and lime-washed, and the stalls roomy. No deposit of manure should be allowed near the sheds. The bedding must consist of clean straw, peat-moss, sand or sawdust, fresh twice a day. The floor ought to be swept an hour before milking. Plenty of light and ventilation are essential, but the shed should not be draughty ; a temperature of about 50° F. is the most suitable. On no account should the cows be hurried or frightened before being milked.

The cow must be kept clean, and before milking the udder and teats should be gently washed with warm water and dried with a soft towel, and the hands of the milker also washed. The milker must wear a clean overall, and the pail be thoroughly scalded out with boiling water. The milk should be aerated by pumping in pure air by means of a suitable machine or by pouring it in a thin stream from can to can in the open air. It must then be cooled to 60° F., or under ideal conditions down to 40° F. When sent by rail, it should be placed in milk cans fitted with lids to exclude dust, and put in vans built on the principle of the refrigerator. It would be still better if the milk were cooled, bottled, and sealed at the dairy in the country, and delivered direct from the station to the consumer. The shorter the distance the milk has to travel, and the fresher it is, the better for the infant. Even in towns it ought never to be more than 12 hours old, and no chemicals should be added as preservatives. Borax, boric acid,



salicylic acid, and formalin are the ones chiefly used. Hydrogen peroxide is probably harmless, though it may enable milk to be used which is not properly fresh. To test for boric acid in milk put 20 c.c. in a beaker, add a drop or two of phenolphthalein solution, and then drop in a solution of caustic soda of about normal strength until a faint pink colour appears. Pour the mixture into two test tubes, and to one part add an equal bulk of distilled water, to the other an equal bulk of a neutral 50 per cent. solution of glycerol in water. If boric acid is present, the mixture in the first tube will become more pink, while that in the second will turn pale or white.

Formalin (40 per cent. formaldehyde) is added as a preservative in the proportion of one drop to each ounce. It delays the action of rennet, gastric digestion, and the pancreatic digestion of fibrin and starch. Schiff's re-agent (fuchsine decolourised with sulphurous acid), impregnated with sulphurous acid gas, is used as a test. Precipitate casein and fat, and add the filtrate to the re-agent; it turns pink if formalin is present. Another test is that devised by Manget and Marion. If a few crystals of amidol are sprinkled on slightly diluted normal milk it becomes pink or salmon colour, and canary yellow if formaldehyde is present. Whey containing formaldehyde turns yellow on adding amidophenol.

To test for salicylic acid make an ether solution, evaporate it, dissolve the residue in alcohol, and add ferric chloride; a violet colour is produced.

*Cow's Milk compared with Human Milk.*—It is impossible to prepare from cow's milk a fluid identical with human milk, even though the chemical composition of the two fluids appears the same. The proteins in different milks are not homologous, and their digestibility depends partly on their special bio-chemical characters. Part of the indigestibility of cow's milk is due to the fat, the globules being from 10-15 times larger than in human milk. There is evidence, too, that the sugar is not chemically, physically, and physiologically the same in the two fluids. All observers are agreed that the proportion of protein coagulable by acid (caseinogen) is much greater in cow's milk than in human milk. In some respects it differs in the two fluids, for it is precipitated with greater difficulty by acetic acid, and more readily by magnesium sulphate from human milk than from cow's milk. The curd formed by the addition of acid or rennet ferment to human milk is finer and more flocculent, and more readily digestible. According to Biedert, cow's milk coagulates in the stomach like human milk, if the ratio of fat to protein is the same. Schlossmann states that the coagulum is the same, if albumin is added. It is largely a question of dilution for, if from 4-5 times its bulk of water is added to cow's milk, the curd obtained on adding dilute acetic acid closely resembles that of human milk. This implies that there is 4-5 times as much caseinogen in cow's milk as in human milk. If it is sufficiently diluted and agitated, the curd with rennet is similar to that of human milk. The following table



shows the relative proportions of the two proteins, and the average differences between cow's milk and human milk. In human milk the caseinogen bears to the albumin a proportion of about 2 to 3; in cow's milk, about 4 to 1. My own analyses gave the high proportion of albumin 1·4, caseinogen 2·6 in cow's milk.

	Human Milk.	Cow's Milk.
Protein .. .. .	1·2	3·5-4·0
Caseinogen .. .. .	0·6-1·3	2·5-3·7
Albumin .. .. .	0·5-1·2	0·5-1·0
Fat .. .. .	3·4	3·5-4·0
Sugar .. .. .	6·7	4·4

In order to prepare from cow's milk a mixture as nearly as possible identical in composition with average human milk, we must start with an average standard for each. For practical purposes the following is a useful and simple standard table :—

	Cow's Milk.	Human Milk.
Protein .. .. .	4·0	2·0
Fat .. .. .	4·0	4·0
Sugar.. .. .	4·4	6·6
Salts .. .. .	0·6	0·2
Water .. .. .	87·0	87·2

The effects of diluting milk with water can be easily calculated from this table. Sometimes it is essential to dilute it with 4 or 5 times its bulk of water, or even more, in order to enable the baby to digest it. This is due to relative excess of caseinogen in cow's milk compared with that present in human milk, and is further proof that there is from 4-5 times the amount in cow's milk.

Of the numerous methods of devising a milk mixture for an infant the following is by far the simplest. Once the initial difficulty of obtaining a superfatted milk of definite fat percentage is overcome, it is easy to devise a suitable weak mixture and gradually to increase its strength. The top half of a good mixed milk which has stood from 4 to 6 hours will contain about the following percentages : protein 4, fat 8, sugar 4. A mixture of 10 drachms of milk sugar, lime water 1 oz., and water up to 20 oz., will contain 6·25 per cent. of sugar. If there be taken of the top milk amounts increased gradually from 5 oz. to 10 oz., and of the sugar solution sufficient to raise the total amount to 20 oz., the percentage composition of the first mixture will be, roughly, protein 1·0, fat 2·0, sugar 6·0, and of the last, protein 2·0, fat 4·0, sugar 5·5. If a higher proportion of fat is desirable,



the milk must be allowed to stand a similar time, and only the top third used. The apparatus required consists of an ice box, 2 syphons, pasteuriser, thermometer, feeding bottles, bottle brushes, absorbent and non-absorbent wool, straining muslin, mixing pitcher, 8 oz. and 20 oz. graduated measures, bottle warmer, rubber nipples, bicarbonate of soda and boric acid.

The home modification of milk carried out in this manner makes it unnecessary to know the exact percentages of fat and protein in the milk used, provided always that the same amount of top milk is taken after the same period of standing. From the effect on the child, as judged by the digestion, character of the stools, and gain in weight, it is easy to decide whether the food should be made richer in protein or fat, or both.

*Laboratory methods.*—To secure accuracy of composition, and to minimise the chances of food contamination, milk mixtures are made up in milk laboratories by various companies, in much the same way as medical prescriptions are made up by chemists. A prescription is sent stating the percentages of proteins (caseinogen and albumin), fat, and sugar required, the degree of alkalinity, the number of feeds and the quantity in each, and the temperature to which it is to be heated, e.g., pasteurised at 158° F. or 167° F. The component parts of the food are derived from the milk of cows, sometimes tested with tuberculin to ensure freedom from tuberculosis, and properly cooled down. The cows are kept under the best conditions of diet and hygiene, and special precautions are adopted during milking for the sake of cleanliness, and in keeping the milk at a low temperature subsequently and free from contamination. Cream of known percentage of fat, fat-free milk, whey, 20 per cent. milk sugar solution, lime water, and distilled water are the materials used. This method saves both doctor and nurse the trouble of writing down accurate directions and carrying them out. It is merely necessary to start with a weak mixture and gradually increase its strength according to the results. This system of feeding has had a very thorough trial, and has not proved satisfactory, except for temporary purposes. Some babies do badly on it, possibly because of too low a percentage of protein, too high a percentage of fat, or the continued use of cooked food. Pasteurisation is strongly condemned by many physicians. Occasionally injurious organisms get into the mixtures in spite of the care taken. The child's stomach is not a test tube, nor is feeding merely a chemical experiment. The strongest argument against percentage feeding lies in the fact that nature does not provide milk of unvarying chemical composition. It has been shown that milk varies at different times of the day, in either breast, and at different stages of each nursing. Consequently, it is scientifically unsound to keep rigidly to definite percentages of the various constituents, and still more so to provide the same percentages in each feed. The constant repetition of meals identical in flavour, quality, and quantity is entirely opposed to nature's method of feeding.



*Artificial Feeding.*—Artificial feeding may be necessary from birth or not until the commencement of weaning, which should normally take place in the tenth month of life, partial suckling being continued for 4 or 5 weeks longer. Weaning may be delayed if the child is debilitated, and during or after acute illness. It is better to wean during cold weather than when it is hot, and not to begin when a child is actually teething. In sudden weaning the child is taken entirely from the breast and put on a milk mixture suitable for its age. It may refuse the food for some hours, or even a whole day, but will give in to the cravings of hunger, if the mother perseveres. Gradual weaning is a better method, as by this means the child's stomach and digestion become accustomed to the artificial food, and it is easier to find out the particular mixture which is suitable. During the first week one such feed is given at 8 a.m.; in the second week a second bottle is given at 8 p.m.; in the third week a third bottle is given at 2 p.m., or 4 bottles are allowed daily; and in the next week or two the remaining breast feeds are also replaced by the bottle feeds. The composition of the food and the amount for each feed depends on the age of the child.

In artificial feeding we have to take into consideration (1) The size of the child's stomach. The age and weight of the child. (2) The quantity of food for each feed. (3) The number of feeds to be given in 24 hours. (4) The composition of the substitute food. (5) The mode of preparation of each feed. (6) The kind of bottle to be employed. (7) The temperature of the food and the mode of administration. (8) The preservation of the food. (9) The cleanliness of all the apparatus.

The size of the child's stomach varies at different ages, in different children of the same age, and is more or less proportionate to the size of the child. The difference in weight before and after nursing represents the weight of food taken, but is not an accurate measure of the capacity of the stomach, for some of the food passes into the intestine during feeding. Estimations of gastric capacity must be based on the results of several such weighings and not on one alone, because of the variation in the child's appetite and the amount of milk obtainable. The gastric capacity varies in different infants of the same size, and only a rough average can be ascertained. *Post mortem* measurements are unreliable, for the stomach is likely to be over-distended. The stomach is very small at birth, and increases rapidly during the first two months of life, and only slowly afterwards.

The stomach capacity may be estimated as equivalent to 1 per cent. of the original weight of the child at birth, and an increase of 1 gm. allowed daily up to the end of the first month. Thus a child, weighing  $6\frac{1}{2}$  lbs. (3,000 gms), would require for each feed during the first week 1 oz.; the second week  $1\frac{1}{4}$  oz.; the third week  $1\frac{1}{2}$  oz.; and in the fourth week  $1\frac{3}{4}$  oz. A larger child might have an extra drachm or two in each feed. In the second month the gastric capacity amounts to  $2\frac{1}{2}$  oz.; for



the next four months to 3-4 or 5 oz.; and from six to nine months to 5-6 oz., and occasionally more.

The quantity of food is based upon the gastric capacity. A bulky food is not necessarily a nutritious one, and too rich food may give rise to digestive disturbance. A child lives and grows by the aid of the food it digests and assimilates. Hand-fed infants are often overfed as to quantity or quality of food; or both may be in excess. In healthy infants the weight of the child is a better indication of its needs than is its age, but neither age nor weight alone is sufficient guide. A wasted infant may require a much larger quantity of food than its weight indicates. On the other hand fat, rachitic infants are unduly heavy, but do not require more food in consequence. The best indication that the food is satisfactory in quantity and quality is a regular weekly gain in weight, combined with contentment, satisfactory stools and the usual indications of health. Overfeeding is indicated by irritability, constant crying, indigestion, vomiting, colic, and unsatisfactory stools.

The artificially-fed child requires the same number of feeds and at the same intervals as the breast-fed. It certainly should not be fed more frequently. The breast-fed child empties its stomach in  $1\frac{1}{2}$ -2 hours, while a bottle-fed one generally requires  $\frac{1}{2}$  hour longer, according to the size and the quality of its meal. The child should have 10 feeds at intervals of 2 hours during the first month; 8 feeds at intervals of  $2\frac{1}{2}$  hours during the second month; and 7 feeds at intervals of 3 hours subsequently. Often after the age of 6 months, and occasionally before, the number of feeds can be reduced to 6 and sometimes to 5 per diem. Prolonged intervals between meals should not be encouraged, because the consequent hunger leads to the food being taken too rapidly, and may result in vomiting or gastro-enteric disturbance.

Cow's milk is the usual food used as a substitute for breast-milk. It should be obtained fresh twice a day. In exceptional cases it may have to be diluted with 7 or 8 times its bulk of water, in order to gradually accustom the child to digest it. It is rare for any child, unless extremely marasmic or suffering from gastric disturbance, to be unable to digest cow's milk properly diluted and given in suitable quantities at the usual intervals. It is a common practice to increase the strength of the food with the increasing age of the child. This is theoretically unsound, for the maternal milk supply increases in quantity, and not in quality, as her infant gets older. At the age of 2 months it is generally possible to train a child's stomach to digest equal parts of cow's milk and water—that is, a mixture containing about 2 per cent. of protein. In strong, healthy children, who have reached the age of 6 months and are taking 6 oz. feeds, and appear to require a richer food or more in quantity, it is better to increase the strength of the food rather than the bulk. The advantages of undiluted milk were urged by Parrot in 1887, and supported by Budin



and Chavane, who obtained very satisfactory results by feeding infants on sterilised, undiluted cow's milk. Prolonged heat makes milk less coagulable by rennet, and thus counteracts the effect of excessive protein in the stomach; but there is no reason to suppose this excess of protein is advantageous to the infant. Certainly such a diet sometimes sets up troublesome digestive disturbance. Satisfactory mixtures for artificial feeding can be prepared according to the simple method above described (p. 50), or as is shown in the following tables, which have been found suitable for the average child, though necessarily requiring modification for special cases. Gravity cream, 16 per cent. fat, should be used in preparing these mixtures. If the rich centrifugal cream containing 40-50 per cent. of fat is used only one-third the quantity must be added.

*Diet during the First Month.*

Week of Life.	One.	Two.	Three.	Four.
Milk .. .. .	2	3	4	5
Cream .. .. .	1	1	1	1
Water .. .. .	5	6	7	8
Lime Water .. .. .	1	1	1	1
Milk Sugar .. .. .	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$

*The quantities are given in drachms.*

*Diet after the First Month.*

Month of Life.	Two.	Three to Six.	Six to Nine.
Milk .. .. .	1 oz.	$1\frac{1}{2}$ -2 oz.	$2\frac{1}{2}$ -3 oz.
Water .. .. .	1 „	$1\frac{1}{2}$ -2 „	$2\frac{1}{2}$ -3 „
Cream .. .. .	1 dr.	2-4 drs.	2-4 drs.
Lime Water .. .. .	3 „	4 „	4 „
Sugar .. .. .	1 „	1-2 „	2 „

After the second month thin barley water can be used instead of water, and the lime water omitted. If cream is omitted the top half of milk after standing should be used.

In preparing each feed the quantities of cream, milk, and water required for 12 hours must be measured out in a graduated measure glass. Ordinary spoons are unreliable for they differ considerably in size. The



necessary milk sugar or cane sugar is dissolved in the water, and the whole thoroughly mixed. The mixture must be kept in a clean jug or tightly sealed glass jar in a cool place, surrounded by ice water, or in a refrigerator. The amount for each feed is taken out, after thoroughly stirring with a clean glass rod. It is poured into a milk saucepan and heated until it just begins to boil. It is then cooled down and given to the child, lime water being added if required. Another plan is to prepare the quantity necessary for 24 hours, divide it up into the number of feeds, put them in separate bottles, and sterilise in a suitable sterilising apparatus, such as Soxhlet's or Hawksley's. After sterilisation the bottles, stoppered with cotton wool, are kept in a refrigerator or cool cellar and used as required. The system of boiling milk when it is first received, and allowing it to cool down gradually, merely covered up with muslin to keep off the dust, is of comparatively little value, except for destroying infective organisms. Such milk goes sour almost as quickly as if unboiled, and many deleterious organisms, or their spores, escape destruction and multiply as the milk cools.

The proper kind of bottle to use is a boat-shaped one, as simple as possible, with a glass stopper and valve at one end and a large mouthpiece at the other, for the attachment of a simple rubber teat which can be turned inside out for cleaning. Bottles with long rubber tubes must never be used, because of the difficulty of keeping the tubes clean; even glass tubes are objectionable for the same reason. A further advantage of the boat-shaped bottle is that each feed must be supervised by the mother or nurse.

The temperature of the food must be about 100° F., ascertained by the thermometer, and not by taste or dipping in the little finger. Neglect of this point is a common source of failure in artificial feeding. Frequently the food is allowed to get cold before it is finished. The food can be warmed by standing the bottle containing it in a vessel half full of hot water, or by means of one of the food warmers in the market.

The child should be fed while half reclining on the nurse's lap, with the head and back supported. The food must be given slowly, and the teat kept full, to prevent air being swallowed. Each feed should occupy from 10 to 20 minutes. The hole in the teat must be of sufficient size to allow the food to pass through slowly, without undue exertion in sucking. After being fed, the baby's mouth is gently wiped, especially the corners, and the child put quietly in its cot and allowed to sleep. Sucking at an empty bottle, or at one of those abominations known as "Job's Comforters," should not be permitted; the latter may lead to dirt or organisms being conveyed to the child's mouth, and both are sources of flatulence and indigestion.

All milk should be fresh and kept in sweet smelling, cool places, such as a cellar or refrigerator, or in a tightly corked bottle in cold or iced water. It must not be kept in a nursery or bedroom, or where it can



be contaminated by smells from drains, housemaid's sinks, or decomposing food. A window ledge with a north aspect is often a convenient place.

Each bottle, after use, must be washed out thoroughly in hot water and then in soda and water, a bottle brush being used. It is then rinsed out several times in clean boiled water and put in a basin of clean cold water or boric acid solution, one drachm to the pint, until next required. Before use it is again rinsed out in pure boiled water. The teats are thoroughly cleaned and kept in a similar way. All the surroundings of the child—the nursery, the cot, and the nurse—must be kept scrupulously clean.

**Bacteriology.**—Fresh milk possesses a certain inhibitory power over the growth of organisms. According to Myer Coplans (1907) breast-milk inhibits bacterial growth absolutely for one hour and almost absolutely in the second hour, the milk being digested in the stomach in the meantime. Growth is, therefore, inhibited both by milk and gastric juice. New cow's milk possesses inhibitory powers for 6 hours, but during the next 6 hours bacterial growth rapidly takes place. The only alternative to breast-milk in preventing infantile diarrhoea is new milk, kept at 32° F. immediately after milking. Even at the end of 24 hours half its inhibitory power remains; while at blood heat this power only persists for 1-2 hours. Under ideal conditions cow's milk has only one-fourth of the inhibitory power of human milk. Preservatives damage this property as well as restrain microbial growth. It is completely destroyed by boiling. Hence, the growth and destruction of organisms in the stomach depend partly on the inhibitory properties of the milk and partly on the gastric juice.

Milk, as it reaches the consumer, contains organisms varying in number from  $\frac{1}{2}$  to 10 or more million per c.c., according to the external temperature, the cleanliness and purity of the milk, and the care devoted to its management. In America certified milk can be obtained containing not more than 30,000 germs per c.c., 4 per cent. fat, and 0.02 per cent. acidity. The longer milk is kept the greater is the liability to changes due to microbial growth and the further is it removed from its character as a living fluid.

The chief groups of organisms are those that give rise to lactic acid, butyric acid, and peptonisation. Lactic acid fermentation is the common cause of milk turning sour, and is due to numerous types of lactic acid bacilli. The degree of acidity is a fair test of the number of organisms present. The lactic acid bacilli are comparatively harmless, and beneficial by crowding out and preventing the growth of other organisms. Sour milk is obviously no longer fresh, and to delay the process of souring, by the addition of preservatives, pasteurisation, or other methods, simply gives other and more dangerous organisms a better chance of development. Sour milk does not go putrid for a long time. There is also evidence that lactic acid bacilli and lactic acid prevent the growth of injurious intestinal flora and act as intestinal antiseptics.



The organisms of diphtheria, cholera, specific fevers, and tuberculosis are readily destroyed by heat, as in pasteurisation, boiling, and sterilisation. In pasteurisation milk is heated to 158° F. for 20-30 minutes. Boiling raises it to a temperature somewhat above that of boiling water; and in sterilisation it is subjected to the influence of superheated steam for 20-30 minutes. Apparently heat will destroy organisms, but not the products of the organisms; so the fresher the milk is when it is heated the less is the risk of the presence of toxins. Unfortunately, though these temperatures destroy organisms, some of the spores escape destruction; for instance, the spores of the *bacillus subtilis* are not destroyed by 6 hours' exposure to a temperature of 212° F., and those of the *bacillus enteritidis sporogenes*, a cause of infective diarrhœa, are not destroyed by more prolonged exposure at an even higher temperature.

Pasteurisation is generally a measure of safety, and does not seriously change the character of the milk, nor affect its taste and smell. Rapid cooling after exposure to the heat is an important part of the process. Commercial pasteurised milk is often an unsafe article of diet.

Boiling alters the taste and smell of the milk, rendering it objectionable to many infants. During cooling a thick, tenacious scum forms on the surface; it is composed of casein, albumin, fat, and salts. The milk is devitalized, and is possibly more indigestible and less nutritious.

Sterilisation should be carried out as soon as the milk is received, by means of one of the various sterilisers in the market. Properly prepared commercial sterilised milk is better in so far as dirt and foreign bodies are removed by the separator, and the milk is fresher when it is sterilised. The risk is that old, unsound or unclean milk, imperfectly sterilised, may be sold under this name. It is far removed from fresh milk, and liable to give rise to scurvy from prolonged use.

Heat has important effects upon milk. Odours are expelled. About 90 per cent. of the carbonic acid gas and 50 per cent. of the oxygen and nitrogen are driven off. Consequently the acidity falls and earthy phosphates are precipitated. Some of the citric acid is precipitated as tricalcium citrate. Ferments are destroyed, salts changed, and other alterations in the milk render it a purely artificial food unlikely to maintain perfect nutrition. These changes begin at 140° F. The milk is not rendered absolutely safe from bacterial contamination, for Flügge has shown that the spores of 12 different kinds of aërobic bacilli survived in milk kept at boiling point for 2 hours; these organisms are liable to give rise to diarrhœa. The antiscorbutic quality of the milk, highest when it leaves the udder, diminishes with the age of the milk, deteriorates with exposure to heat, and may be absolutely destroyed. Possibly this quality is connected with the citric acid present in milk. Heat is also said to render fat assimilation more difficult, to partly destroy sugar, to convert organic phosphorus into inorganic phosphates, and to destroy nuclein



and lecithin. On the whole it is probable that no cooked milk is as nutritious as uncooked milk, and that the nutritive value diminishes in proportion to the degree and duration of heating.

It is unsafe to use unheated milk, except under special circumstances and in cold weather. On the other hand, no amount of heat will destroy toxins previously formed, nor will convert bad milk into good milk. A clean milk supply is of more importance than methods employed to counteract the defects of imperfect milk. Unfortunately such measures are at present necessary. Hospitals should insist on a supply of pure clean milk from healthy cows, cooled down to at least 60° F., unadulterated, not pasteurised, free from preservatives, and containing 3.25-3.50 per cent. of fat.

It is sometimes important to be able to tell whether milk has been cooked. Some dairies send out their milk pasteurised. Fresh unheated milk turns red on the addition of guaiacol and 1 per cent. solution hydrogen peroxide, added drop by drop. Saul's test consists in adding 1 per cent. fresh aqueous solution of ortol and one or two drops of peroxide to 10 c.c. of milk. If fresh, the colour becomes a vivid red. Milk heated above 158° F. does not give these peroxide reactions; there is no change in colour.

**The Effects of Heat and Attenuants on Curdling.**—By heating milk to various temperatures up to 120° C., for periods of  $\frac{1}{4}$ -1 hour, and then testing the coagulability with acetic acid and rennet ferment, the temperature of the milk being first reduced to 40° C., the following results were arrived at. It was found that heat alone did not render cow's milk similar to human milk in its reaction to acetic acid. If the exposure at a high temperature was prolonged, the curd became more coherent. If the milk were diluted with water, before being heated, the size of the curd depended on the degree of dilution. The finest curd, very like that of human milk, was obtained by dilution with two parts of water and exposure for  $\frac{1}{4}$  hour at 100° C. Using weak barley water, thick barley water, and lime water, similar results were obtained as regards the degree of dilution. The finest curds were obtained when thin barley was used; they were even finer than with plain water. Up to 80° C. lime water did not prove as satisfactory a diluent as thin barley water. Above that temperature the milk was altered in colour by the action of heat on the lime water, and the results were of no value.

One drop of rennet coagulated fresh milk in two minutes, but if the milk were thoroughly sterilised by exposure to 100° C. on 3 successive days for 1 hour daily, it required a large quantity of rennet to produce coagulation. Even at the end of 45 minutes only a soft curd was produced. More rennet is required to curdle heated milk, and the amount increases with the height of the temperature and the duration of the exposure. If the milk were diluted, as well as heated, the softness and degree of coherence of the curd depended chiefly on the degree of dilution, rather than on



the amount of heat applied. Barley water did not appear to have any more effect than plain water on rennet curdling, but the addition of alkalies lessens this coagulability.

These experiments tended to show that the essential factor in rendering cow's milk like human milk consists in simple dilution with water to such an extent as to reduce the percentage of casein to that present in human milk. They also clearly prove that heat modifies caseinogen, possibly by action on the lime salts, to such an extent that it is less coagulable by rennet. Alkalies produce a similar effect, e.g., the addition of a grain of citrate of soda to each ounce of milk. More may be added. It is generally stated that citrate of soda precipitates some of the lime salts; but, according to Variot (1905), it rather increases their solubility. Citrate of soda is a neutral salt, very soluble in water, cheaper and more effectual than citrate of potash. A drop or two of chloroform should be added as a fungus is liable to grow in weak solutions. It appears probable that the beneficial effect of adding citrate of soda, or alkalies, to milk is the same as that produced by sterilisation. By preventing the coagulability with rennet ferment, the milk does not curdle in the stomach, and passes on readily into the small intestine. Gastric digestion is relieved and replaced by pancreatic digestion.

**Subsidiary Methods of Feeding.**—Mechanical attenuants include decoctions of barley, oatmeal and rice. Egg albumin, gelatine and dextrinized foods are sometimes used. Oatmeal is the richest of the cereals in fat, and is somewhat laxative. Rice is practically devoid of fat. Sometimes the milk, whole or separated, is curdled, and the curd broken up mechanically with a fork and squeezed through muslin or a fine hair sieve, thus breaking up the casein particles, which do not again become blended into coherent masses in the stomach. For curdling the milk, junket tablets or various preparations of rennet can be used. The latter are variable in strength, made with glycerine, and may not be pure. Beauchamp Hall recommends the use of tablets containing purified and standardised rennet sufficient to curdle  $\frac{1}{2}$  pint of milk. Each tablet is made up with sod. bicarb. gr.  $1\frac{1}{2}$ , calcii. phosphat. gr.  $\frac{1}{2}$ , and lactose gr. 5. Ten ounces of milk yield seven of whey.

**Whey.**—As the curd contracts it squeezes out the fluid portion or whey. This is consequently devoid of casein, practically fat free, and contains about the same amount of sugar and salts as the original milk. It contains on an average 0·85 per cent. of albumin. If the curd is squeezed through muslin, some of the casein and fat globules pass through, and the fluid is thereby enriched. Whey is an easily digested, weakly nutritive fluid, useful as a temporary food and in the early months of life. Used as a diluent of milk, it is a valuable means of increasing the amount of albumin, without increasing the caseinogen. A mixture of cream and whey is more valuable, in that it contains much more fat. The addition of



1 dr. of cream, 16 per cent. fat, to 2 oz. of whey is equivalent to adding 1 per cent. of fat. When either cream or milk is added to whey, the whey must be first heated to a temperature of 150° F. to destroy the rennet ferment. This is not absolutely necessary when only small quantities of cream are added, or if rich centrifugal cream is used.

*Tartrated whey* is recommended by Still and Myers as a cheap substitute. Eight grains of tartaric acid are added to milk which has been brought to a boil, and it is then kept simmering for 5 minutes and is strained through butter muslin. Its specific gravity is 1030; protein 0.58 per cent., fat 1.2 per cent., and it is only faintly acid to litmus. It can be added to milk without causing further curdling.

Still also advises the use of a cheap cooking sherry, about 1s. per bottle, in the making of *White Wine Whey*, because it contains more acetic and tartaric acid and less alcohol than the better wines. Ten ounces of milk are brought to a boil, 2½ oz. of sherry added, and heat again applied until the mixture begins to boil up. It is then removed from the fire and allowed to stand 3 minutes and strained through butter muslin. One ounce of this whey is about equivalent in alcoholic strength to 25 drops of brandy.

*Citric Acid Whey* is made by boiling a pint of milk with 2 teaspoonfuls of lemon juice.

*Peptonised or predigested milk* is prepared by means of Fairchild's *zymine* powders, or the Allenbury peptonising powders. Directions for making it are given with the powders. The milk is diluted with one-fourth part of water, and the duration of peptonisation varies from 10 to 20 minutes, after which the mixture is boiled to prevent further digestion. If the process is carried on until peptonisation is more complete, the mixture becomes bitter. Curdling during peptonisation is probably due to the use of stale powders. Fairchild's peptogenic milk powder is also used for this purpose. It consists of pancreatin, bicarbonate of soda, and milk sugar. A mixture of milk 2 oz., cream 1 oz., water 2 oz., and peptogenic milk powder 1 measure, is heated slowly, with constant stirring, so that it is brought to a boil at the end of 10 minutes; or is heated quickly to 100-140° F. for 6 minutes. The resulting fluid is often called *humanised milk*. According to Leeds' analysis it contains protein 2.0, fat 4.5, sugar 7.0 per cent.; and the proteins are in a minutely coagulable and digestible form. Partial predigestion does away with the need of other methods of attenuating the curd, though it may be advisable to lower the percentage of proteins by dilution with water. By gradually reducing the duration of the exposure to heat, the amount of predigestion can be reduced and a return to the ordinary milk diet effected. Or one feed of unpeptonised milk may replace one of the peptonised, and, if this is digested, the other feeds can be gradually replaced in the same way. Used reasonably and for not too long, say, not more than 3 or



4 months, it apparently does not injure the digestive capacity of the child. It is chiefly valuable for delicate or wasted children with digestive powers weakened or lost; for young infants who cannot digest modified cow's milk; and in febrile disorders interfering with digestion. When ordering peptonised milk full directions must be given about the process of preparation and amount of each feed, and the number of feeds to be given in 24 hours.

Various kinds of *dried milk* are occasionally useful. Some of them are preparations of pure casein, e.g., biogene, casumen, plasmon, protene, and tilia. Casein is combined with ammonia, as eucasin; with sodium, as nutrose; with sodium glycerophosphate, 5 per cent., as sanatozen; with albumose, 20 per cent., as sanose. Dried casein is a nutritious food on a par with cheese made from skimmed milk, and is a valuable addition to a diet deficient in protein.

Desiccated whole milk is sold under the names of glaxo and lacvitum, and desiccated skimmed milk as lacumen. Glaxo contains protein 22·2 per cent., fat 27·4, sugar 41·0, ash 3·4, water 6·0 (Guy's Hospital Gazette). Lacvitum contains protein 28 and fat 29 per cent. Suitably diluted, these preparations are useful as temporary foods in hot weather, but must not be long continued because of their liability to cause scurvy, as well as on account of the deficiency in fat.

*Condensed milk* is widely used, and a valuable food when travelling, in hot weather, in marasmic conditions, and if good cow's milk is not obtainable. There are two main varieties: (1) simple condensed milk, and (2) condensed milk with added cane sugar. They are prepared from skimmed milk, whole milk, or whole milk with added cream, and evaporated down to one-third or one-fourth of their bulk. Unsweetened condensed milk, such as the Ideal, First Swiss, Viking and Hollandia brands, contain protein 8-11 per cent., fat 9-12, and lactose 13-18. Of these the Ideal is the richest in fat. They are useful in the treatment of infantile diarrhoea and other alimentary disorders in which a large amount of cane sugar is contra-indicated. They must be diluted with water, and cream and milk sugar added in order to bring their composition up to that of human milk. The milk soon turns sour when the tin is opened.

Of the sweetened condensed milks, Nestlé's and the Milkmaid brands are reliable, and are prepared under satisfactory conditions of purity and general cleanliness. Similar brands are the Rose, Full Weight, Anglo-Swiss and Peacock. All these contain approximately protein 10, fat 10, sugar 60 per cent. They must be diluted with eight times their bulk of water, often more, and the addition of cream is necessary to raise the percentage of fat. Condensed milk is given much too diluted, and is popular on account of its easy digestibility and absorption, and because it is so easy to prepare. Its weakness is compensated for by the greater quantity taken, but the usual defects arising from insufficient protein



and fat and excess of sugar are likely to occur (pp. 31-32). Babies fed on condensed milk almost always develop rickets. Often they are very fat, are liable to bronchitis and gastro-intestinal derangements, and possess little vital resistance to disease. Used under medical advice, excellent results are obtained, and the lives of many marasmic infants are saved by this diet. Extra protein can be given in the form of egg albumin, fat in the form of cod-liver oil emulsion, and the tendency to scurvy counteracted by means of fruit juice. Only those milks condensed from whole milk, or from whole milk with added cream, should be allowed in infant feeding, and even they must not be continued as a permanent diet for more than a few months.

*Ass's milk* is very useful as a temporary food, and can be obtained from Welford's Dairy Company, in London; but is somewhat costly, 3s. per pint. It is supplied in sealed glass bottles. One ass supplies enough milk daily for three infants under 3 months of age, two during the fourth and fifth months, and one only after this. The milk is simply warmed to a temperature of 100° F. The composition is shown in the following table:—

*The Composition of Ass's Milk.*

	Dujardin- Beaumetz.	Ellenberger.	Cheadle.	Wynter Blyth.	Peligot.
Protein ..	1·23	1·6	1·7	1·89	1·95
Fat ..	3·01	0·9	1·4	1·02	1·02
Sugar ..	6·93	5·6	6·4	5·50	6·48

According to most analyses it is very deficient in fat, but contains about the same amount of both kinds of protein and of sugar as human milk. Babies do well on it for a short time only. It must not be given for more than two or three months, and it is an advantage to add cream to it. Its laxative properties are counteracted by lime water and destroyed by boiling.

*Goat's Milk* is said to be more digestible than cow's milk, but judging by various analyses it is closely allied to it. Sometimes it has a strong odour and unpleasant taste, if the animal is ill, fed on garlic, ivy, &c., or milked into dirty pails. Its chief advantage lies in the fact that any one with a small plot of grass can keep a goat, feed it carefully and obtain fresh pure milk.

*Kephir*, *Matzoon* and *Koumiss* are modifications of the milk of the cow, goat, mare, or ass, prepared by fermentation by means of yeast or Kephir grains. They are quite unnecessary. It is preferable to give



alcohol, when required, in simpler forms and more accurately measured doses.

*Buttermilk* is useful. It is made from sour cream or milk, and should be less than 24 hours old. It contains protein 2-3, fat 0.25-1.5, sugar 3.0-3.5 per cent., the decrease in sugar being due to the lactic acid fermentation. Its acidity is about 0.5 per cent. and calorie value 9-12 per ounce. In the process of preparation the calcium casein is transformed into calcium lactate. It should be made from sour milk previously skimmed. One pint is churned in a glass churn, holding one quart, for 15 minutes, and at a temperature of 60-70° F. It is not often taken raw, but usually sweetened and sterilised. Add 1 teaspoonful of wheaten flour and 4 of granulated sugar to a quart of buttermilk, with constant stirring, and heat to boiling point in a double saucepan. If heated too much it will curdle, unless constantly stirred or made slightly alkaline by the addition of carbonate of soda. It must be cooled rapidly and kept in bottles. It contains numerous lactic acid bacilli, if unheated, and a considerable amount of lactic acid. A condensed buttermilk, "Nutricia," can be obtained.

*Lactobacilline Milk* is analogous to buttermilk, and is prepared from milk by inoculating it with huge numbers of lactic acid bacilli. It is then sweetened and sterilised, or given unheated if it is desired to give large quantities of lactic acid bacilli as well as lactic acid. These foods are useful in enteritis and colitis, and are supposed to limit intestinal putrefaction.

**Proprietary Foods.**—In most of the proprietary foods there is a large quantity of carbohydrates, frequently in the form of starch. In some the starch is partially changed into dextrins, dextrose, and maltose. Others contain much cane sugar. Many have a basis of condensed milk. Seeing that there is no substance like starch in milk, it is unlikely to be a suitable food for young infants. The amylolytic ferment in saliva is secreted in very small quantities in the early months of life. It is found in the sub-maxillary and parotid glands, and in the pancreas of newborn infants, even if premature. Possibly a small amount of starch can be digested by this ferment, as well as by heat and moisture, even in the youngest infant; but salivary secretion is very scanty and rarely appreciable before the age of 2 or 3 months. During the third month secretion is much more rapid, and its activity fully develops by the end of the first year. The action of the ferment depends partly on the variety of starch, and is greater when the starch is boiled. The pancreatic secretion exerts a much more powerful action. We may conclude that a weak solution of starch can be used as a diluent after the second month of life, but that any excess may be injurious, because undigested starch is liable to fermentation. Many infants do better on milk and water than when barley is used as a diluent, and it is often advisable not to commence the use of starch before the age of 6 months.



Many objections can be raised against the use of proprietary foods in infancy. A child can be brought up on suitably modified cow's milk. Extra fat can be given in the form of cream, olive oil, cod-liver oil, butter, or margarine. Extra protein can be given by using whey or egg albumin water as a diluent, or by adding one of the casein preparations. Carbohydrate can be added in the form of milk sugar, cane sugar, maltose, or honey. After the age of 6 months the common starchy foods are at our disposal. An excess of carbohydrates in the foods containing altered starch produces fatness rather than health and strength. The mortality among infants brought up on artificial foods is enormous. Some infants digest these foods with impunity, and even with advantage, but they are exceptional. The magnificent babies of manufacturers' advertisements only exist in imagination, or, when seen in the flesh, are simply fat and rachitic. Rickets and scurvy are undoubtedly often due to the use of these foods. Fortunately, their disadvantages are reduced by giving them mixed with cow's milk.

Many proprietary foods prepared from cow's milk only have been already considered. The next group consists of those prepared from cow's milk with the addition of carbohydrates, and include :—(1) *Condensed milk with malted flour*, e.g., Allenbury No. 2 Food, Horlick's Malted Milk, and John Bull No. 1 Food. These are practically devoid of starch. (2) *Condensed milk with partly malted flour*, as in Manhu food, Milo food, and Theinhardt's Infantina. (3) *Condensed milk with unchanged flour*, as in Anglo-Swiss, Franco-Swiss, and American-Swiss food. The foods in group (1) only differ from sweetened condensed milk in containing maltose instead of cane sugar. Those containing starch are unsuitable, except in very small amounts, before the sixth month of life.

*Benger's Food* must be included in this group, for it is a mixture of cooked wheaten meal and extract of pancreas, and is prepared with milk. The ferments partially peptonise protein and partially convert the starch into soluble dextrans and sugars. The fat is unaffected. It is valuable for marasmic infants after the sixth month of age, and can be used in small quantities of  $\frac{1}{2}$ -1 teaspoonful in each feed as early as the fourth month of life. The addition of a small half teaspoonful to each feed is about equivalent to using thick barley water, instead of water, as a diluent, and has the additional advantage of partly digesting the food. Care must be taken that the food is not increased in quantity at the expense of the essential cream and milk. *Carnrick's Soluble Food* is made of desiccated milk, malt and wheat flour, lactose, and extract of pancreas. It is comparable with the foods made from condensed milk and partly malted flour, and is much inferior to Benger's Food, seeing that it is not made with fresh milk.

The next group consists of the pure *Carbohydrate Foods*, and may be subdivided into three classes :—



(a) *The Starch practically unchanged*.—Neaves', Ridge's, Opmus, Albany, Robinson's Patent Barley and Groats, Scott's Oat Flour, Chapman's Flour, &c.

(b) *Partly Malted Foods*.—Allenbury No. 3, Cheltine Infants' Food, Coomb's Malted Food, Hovis No. 2, John Bull No. 2, Moseley's, and Savory and Moore's Food.

(c) *Completely Malted Foods*.—Mellin's, Horlick's, Paget's Malted Farina, and Hovis Babies' Food. Others of a like nature are Cheltine Maltose Food, Diastased Farina, and Racia.

The completely malted foods can be given at an early age in small quantities and in preference to cane sugar, provided that they are not unduly laxative and are not given instead of cream and milk. It may be advisable to use the partly malted foods before the end of the sixth month, but those composed of unchanged starch are never really necessary. If it is desired to gradually train the child in the digestion of starch, we can begin with a purely malted food, as Mellin's, pass on to a partly malted one, such as Savory and Moore's, and then give one of unaltered starch. It is simpler and generally quite as satisfactory to begin with thin barley water and gradually make it thicker and thicker.

The *Meat Preparations* include various brands of meat teas, extracts, juices and jellies, peptones and peptonoids, and meat powders. Most of them consist of extractives, of no nutritive value, possibly stimulating the digestive functions and throwing an extra strain on the liver and kidneys. The high proportion of salts and extractives is liable to cause thirst and diarrhœa. Some of the meat jellies, though expensive and innutritious, have a pleasant flavour, are taken by a feverish infant who refuses other food, and may encourage appetite. Similarly hot fluids, like beef tea, are pleasant to the febrile tongue and in disordered digestion. In small quantities they do no harm, and comfort an anxious mother who fears her child will die of starvation, and has a profound faith in the nutritive value of these preparations. As ordinary articles of an infant's diet they must be condemned, for they are apt to spoil the appetite for the simpler milk and carbohydrate foods. If given in any quantity they set up an irritable, neurotic state and a tendency to night terrors. There is no serious objection to giving home-made chicken tea, mutton broth, and similar foods in the last quarter of the first year of life, especially if it is desired to reduce the nutritive value of the diet. A daily feed of chicken broth, thickened with pounded vegetables and some cereal, is useful for fat, plethoric, overfed babies, who have been brought up on an excess of malted foods, cream and milk. It reduces the total calorific value of the diet, and gives the stomach less work to do.

Of the proprietary meat preparations commonly used, *Liebig's Extract* is free from protein and fat; *Bovinine* appears to be a mixture of blood and glycerine; *Puro* is apparently chiefly made of egg albumin; *Vinsip*



is a mixture of blood, boric acid, and alcohol; and *Valentine's meat juice* is, according to Chittenden, most innutritious and contains only 0.55 per cent. of coagulable protein and gelatine. *Bovril* appears to be the most trustworthy and nutritious. No meat preparation containing alcohol or kola should be given to infants.

Though not really necessary, proprietary foods are often of very great value, more especially the malted foods, if they are used with discrimination as additions to the diet, and not as substitutes for cream or milk. It is important, therefore, to be acquainted with their composition, or one is likely to be led into errors of selection and fatal dietetic calamities. Unfortunately the evil results of erroneous methods of feeding may not develop for some months, and are constantly ascribed to other causes. A skilled acquaintance with the use of cream, milk, various kinds of sugar, and starchy foods, renders one independent of recourse to patent foods.

**Diet after Weaning.**—In the process of weaning, during the tenth month of life, the diet is changed to one of cream, milk, barley water, and sugar, according to the methods recommended under the head of artificial feeding. Instead of barley water, barley jelly, oatmeal jelly, or a thin gruel made with cornflour, ground rice, fine oatmeal, or arrowroot may be added to the milk and well boiled. The next step is the addition, of one-half to one whole nursery rusk or Robb's biscuit, to one or two feeds a day of milk two parts and water one part, or equal parts. If the child is at all anæmic or rachitic, it is advisable to give from one to two teaspoonfuls of yolk of egg, raw or lightly boiled, in two of the daily milk feeds, or the yolk of egg and raw meat juice on alternate days. The drawbacks to meat juice are that it is difficult to get quite fresh meat, that it keeps badly in hot weather, and the slight risk of the child acquiring tapeworm. It is best prepared by means of a meat press, such as the Hercules meat press, but can be made by mincing meat, pounding it up in a mortar with a pinch of salt, and standing it in a clean cup, with just enough cold water to cover it, for 2-3 hours, and then squeezing it through muslin. It must be kept in a closed bottle surrounded by ice in a refrigerator. This diet can be continued till the end of the first year, or amplified in the last month by the addition of custard or plain milk pudding. Five or six meals a day are sufficient.

*Diet from 12 to 18 months of age.*—The simple milk and carbohydrate diet may be continued, if the child and mother are satisfied. The milk must be still diluted with one-third its bulk of diluent. Often a more mixed diet is desired, and can be drawn up on the following lines:—

*First Meal*, at 6 to 7 a.m. :—

Six ounces of boiled milk, hot or cold.

A slice of stale bread or a rusk broken up and soaked in the milk,  
or a slice of thin bread with dripping or butter.



*Second Meal*, at 8.30 to 9.30 a.m. One of these daily :—

A small basin of bread and milk.

A little fine oatmeal porridge, with cream or milk.

A basin of thick milk gruel.

A cup of thin cocoa made with milk, and a little thin bread and butter.

*Third Meal*, at 1 p.m. :—

*First Course* ; one of the following :—

Mashed baked old potato moistened with milk, chicken or mutton broth, the red gravy of undercooked meat or meat juice (1-3 oz.).

Lightly boiled or poached yolk of egg mixed up with stale bread crumbs or mashed potato and gravy.

Stale breadcrumbs soaked in gravy or meat juice.

*Second Course* ; one of the following :—

A large tablespoonful of custard, tapioca, cornflour, ground rice, or semolina pudding, blancmange or junket.

Cold boiled water, or milk and water, to drink.

*Fourth Meal*, at 5 p.m. :—

The same as the first meal, or thin cocoa with bread and butter.

*Fifth Meal*, at 9 p.m. :—

A large cupful of milk gruel made with rice, sago, tapioca, or hominy, or a rusk or sponge finger soaked in milk.

Many other foods can be given at this age, especially those of a carbohydrate nature, preparations of egg, and small quantities of jellies and fruits, such as baked apple, the pulp of grape and oranges, and even mashed banana if it is thoroughly ripe. If the child is healthy and strong a small portion of pounded-up fish, chicken, or meat may be given at the midday meal, when 15 months old.

During the next period from the eighteenth month to the middle of the third year of life, a somewhat similar diet is given, including meat, fish, vegetables and fruit.

*Diet from the Age of 18-30 months.*

*First Meal*, 6.30 to 7.30 a.m. :—

A large cupful of milk, with rusk, stale bread, or milk biscuit broken up and soaked in it, or some bread and butter.

*Second Meal*, at 8.30 to 9.30 a.m. ; one of the following :—

A basin of bread and milk.

Oatmeal porridge, with cream, milk, or golden syrup.

A basin of hominy grits or Quaker Oats, and milk.

Boiled milk or cocoa, with bread and butter.

Boiled milk, a lightly boiled egg, and bread and butter or dripping.



*Third Meal, at 1 p.m. :—*

*First Course ; one of the following :—*

Mutton, undercooked beef, chicken, turkey, or fish, minced up finely, pounded up in a mortar into a paste for the younger children ; one tablespoonful of one of these mixed up with mashed old potato or stale breadcrumbs and gravy : vegetables, such as spinach, young green peas, young asparagus tips, &c.

Lightly boiled or poached egg, with mashed potato or stale breadcrumbs and gravy.

A large cupful of broth or soup (purée) with mashed potato or stale breadcrumbs.

*Second Course ; one of the following :—*

Custard or plain milk pudding, blancmange, cornflour or ground rice mould, plain sweetened jellies, baked apple and junket.

*Fourth Meal, at 5 p.m. :—*

A large cupful of milk, with bread and butter, rusks, milk biscuits, or sponge fingers.

A cup of thin cocoa, made with milk, and bread and butter or honey.

A small basin of milk gruel or bread and milk.

*Fifth Meal, at 9 p.m., if the child wakes for it :—*

A cup of boiled milk, with a biscuit or piece of bread soaked in it, or a cup of milk gruel.

Other vegetables, such as broccoli, cabbage, turnip tops and cauliflower, may be given, if passed through a sieve. Carrots, potatoes, and artichokes may be allowed in the form of vegetable purées, providing they are thoroughly well cooked. Water should be taken as a drink at dinner time, and either water or barley water given frequently in small quantities between meals to relieve thirst and in hot weather. Tea, coffee, and stimulants may not be permitted.

When all the milk teeth are cut, four meals a day are required, distributed as follows :—

*Diet after the Age of 2½ years.*

*Breakfast, at 7.30 to 8.30 a.m. :—*

Either bread and milk, porridge with milk or golden syrup, or hominy grits or other cereal food and milk ; egg boiled, poached, or scrambled ; bread, with butter, dripping, or fruit jelly ; milk or cocoa to drink.

*After the Age of 4 ; second course :—*

Eggs ; plain omelette ; a little fat bacon or ham ; a little fish ; fresh potted meat.

*Lunch, at 11 a.m. :—*

A cupful of milk, and a slice of bread and butter or a plain biscuit.



*Dinner*, at 1 p.m., of two courses, selected from :—

Vegetable purées, minced beef, mutton, chicken or turkey; boiled fish; poached egg; brains; sweetbread; mashed potatoes and pounded-up vegetables; macaroni. Milk puddings, blancmanges, farinaceous moulds, stewed fruits, jellies.

*Tea*, at 5 p.m. :—

A basin of bread and milk, milk gruel, hominy and milk; a cup of milk with bread and butter, biscuit, or bread and potted meat or fish; fruit jellies may also be allowed.

*Supper*, at 9 p.m., if required :—

A cup of milk and biscuit.

After the age of 4 years meat need not be minced if the child eats slowly and masticates thoroughly. Rabbit and veal must be well boiled, minced, and pounded. Red meat should not be given more frequently than three times a week, because of its stimulating effects on metabolism and nervous tissues. If the diet is plain, and the child eats slowly, it may eat as much as it wants. After the age of 5 years three meals a day are generally sufficient, but in addition milk and biscuit or some fruit may be allowed in the middle of the morning, and more milk and biscuit at bedtime. Eating between meals must not be encouraged, as it leads to loss of appetite. Highly seasoned made-up dishes, fried foods, and indigestible foods such as pork, tough steak, duck, goose, nuts, candied fruits, and pineapple should be avoided. If not too new or decayed, cheese can be given. Salt should be the only condiment. Fruit must be ripe, not over-ripe, and not allowed in unlimited quantity.

#### *Diet during School Life.*

The diet must be liberal in protein and fat, contain a due supply of vegetables, and above all the food must be fresh. The child should have a cup of milk or cocoa, with biscuit or bread and butter, if there is a morning lesson or morning chapel before breakfast.

Breakfast should consist of two courses :—(1) A cereal food, such as porridge, Quaker Oats, etc., with salt, milk, cream, or golden syrup, or bread and milk; (2) eggs in some form, fish, bacon, ham, tongue, meat rissoles, etc.; and fruit, lettuce, or radishes when in season. Cocoa, milk, or weak tea should be given, and a liberal supply of bread, rolls, toast, and butter.

In the middle of the morning milk and biscuit or fruit can be given. This is important in cold weather and for delicate children.

At dinner each child should be given a sufficient portion of meat, potatoes, and green vegetables. Fish may be allowed once a week, preceded by a vegetable purée. Any kind of simple pudding is suitable for the next course, especially those that contain suet, jam, or fruit. In addition, plain cheese, bread and butter should be eaten if wanted. Cheese is a



valuable addition to a diet deficient in protein. Water, barley water, oatmeal water, or lemonade are the usual drinks.

Tea should be given at 6 p.m., and consist of unlimited bread and butter; various kinds of jams or marmalade; freshly potted meat or fish; sardines; egg, cress, or tomato sandwiches; plain cakes and biscuits, with milk, cocoa, or weak tea:—if sufficiently liberal, supper is unnecessary. Older children, who do not go to bed till 10 or 10.30 p.m., should have more protein food at tea, such as fish, meat rissoles, calves' head, or eggs. If not, milk and biscuit may be allowed at 9 p.m., but no late meal of bread, beer, and cheese. Alcohol is quite unnecessary during school life.

Fats, nitrogenous foods, and milk are of the utmost importance. A child requires from  $\frac{1}{2}$ -1 lb. of nitrogenous food daily, and 1-1 $\frac{1}{2}$  pints of milk. Animal proteins must not be replaced by vegetable ones, and no tinned foods are permissible except sardines. The proteins and fats must be distributed, and neither given mainly at only one meal.

Girls require just as liberal a diet as boys, if they get as much exercise. This is especially necessary during the period of rapid growth, which takes place two years earlier in girls than in boys, namely, from 11-16 years of age. Refusal of food indicates a visit to the tuck shop, a hamper from home, lack of variety in the diet, ill-cooked or badly served meals, tainted food or illness.

#### *Diet during Illness.*

So great is the importance of diet in the treatment of disease in children, that it often takes precedence of every other measure. Many ailments depend upon improper food or overfeeding. In the treatment of all gastric and intestinal affections the diet must be carefully regulated; in babies according to the methods already discussed. After the first year of life it may be advisable to precede the treatment by temporary starvation. The diet is of special importance in lung affections, for over-distension of the stomach by food or gas due to fermentation presses up the diaphragm, interferes with the action of the heart, and is often the cause of a fatal issue. The general principles of feeding sick children consist in giving regular meals of suitably prepared food appropriate for the particular age of the child and in quantities suitable to its requirements. The child in bed does not need quite as liberal a diet as one who is actively running about, but unless there are special reasons, such as fever or alimentary disturbance, it may have much the same as a similar child in health. Sometimes it is necessary to give greater variety and predigested foods. In acute illness the digestive capacity is weakened, and it is easier to set up alimentary disturbance than to cure it. Anorexia and vomiting are the infant's method of showing it does not require food. Allow a liberal supply of water in fever. Gavage is sometimes needed.



## CHAPTER IV.

### THERAPEUTICS.

*Nursing—Hydrotherapy—Local Applications of Heat—Counter-irritation—Cupping—Bleeding—Pharmacotherapy—Cutaneous Therapy—Rectal Therapy—Fever—Collapse—Vaccine Therapy—Anæsthesia.*

The treatment of disease is not limited to the administration of drugs, which are by no means always necessary. Indeed, drugs are constantly the least important factors in obtaining good results, though they can be frequently used with the greatest advantage, and are often imperatively needed. Many infantile ailments are cured by dietetic and hygienic measures. Bear in mind that the natural tendency of many diseases is to end in recovery, and that the outlook is often more favourable in the child than in the adult, because of its greater powers of regeneration. Infants, too, in particular, have abnormally sensitive alimentary and nervous systems, rendering their efficient treatment by diet and drugs more delicate and difficult.

**Nursing.**—A sick child, often a healthy one, requires one or two nurses. The doctor is very dependent on the attendants and surroundings, if the full benefit is to be derived from the measures which he advises. The bed should be warm, hot bottles used when needed, and the position of the child one of comfort. Clothing must be warm, light, and non-irritating. The room must be properly ventilated, yet the child protected from draughts. Linoleum on the floor is better than any kind of carpet, for it is easily kept clean. Dusting should be done with a damp cloth, moistened with a disinfectant in infective cases. A sweating child requires the clothes changed, so that it does not get chilled, and the body should be sponged with warm water. Cleanliness of the mouth, teeth, skin, hands, and body as a whole must be ensured. Care must be taken to prevent bed sores by rubbing the skin with a weak spirit lotion, by protecting it from irritation due to crumbs, etc., in the bed, by padding, air cushions, and such-like. Intertriginous ulceration is guarded against by cleanliness, drying with soft warm towels, dusting powders and mild ointments. The exhaustion due to crying can be prevented by kind and tactful management.

**Hydrotherapy.**—Water and soap are used for cleanliness and the prevention of ill-health, due to the inefficient removal of dirt from the



skin interfering with elimination and liable to set up local or general cutaneous mischief. Distilled, rain, or river water is the best. Hard water is disadvantageous, in that it necessitates the use of much more soap, which may perchance be of an irritating variety. Such water can be softened by adding bran or oatmeal, in a bag if preferred on æsthetic grounds. It should not be softened by adding borax, soda, or ammonia, for these chemicals combine with and saponify the fat of the skin, making it harsh and dry. Glycerine and water, or glycerine of starch, applied after washing, keeps the skin moist, and may prevent chapping, but renders the epidermic cells abnormally sensitive. Frequent hot baths increase exfoliation of the skin and favour imperfect cornification, besides being somewhat enervating.

*Soaps* should be superfatted to the extent of 3-5 per cent., and should not contain cocoanut oil. The best soaps are neutral ones made from pure fats. Free alkali in a soap dissolves out the natural protective fat of the skin. Free fatty acids are irritating and readily absorbed. Glycerine or resin, a cheap substitute for good fat, is not to be recommended. Transparency, due to the presence of sugar instead of glycerine, renders such a soap injurious in eczema. Disinfectant soaps contain sanitas, tar, izal, carbolic acid, etc. Therapeutic soaps are made to contain about 10 per cent. of such substances as ichthyol, anthrasol (colourless tar), and balsam of Peru with sulphur.

The *First Bath* is given at a temperature of 100° F., after well oiling the body to remove the vernix caseosa, using a pure castile, Icilma, or Vinolia soap. After this a *Daily Bath* is given; sponging only until the cord has separated. Up to 3 months of age the water should be at 98° F.; then it may be reduced to 95-90° F. during the first year, and 5 or 10 degrees lower in the second year. The bath should be given in a warm room, before an open fire, out of a draught, and for about 5 minutes. The child must be rapidly dried with a soft warm towel, avoiding vigorous friction during infancy, and dusting powder applied. Use little soap, and that a superfatted one. If the bath causes blueness and coldness of the extremities, the child should only be sponged.

*The Bath after the First Dentition.*—A daily sponge bath, at a temperature of 60-70° F., is a useful tonic to the vascular and nervous systems, and diminishes the liability to catarrhal affections. A good large tub or bath is necessary, and a large sponge or flannel. The child is rapidly sponged all over and then quickly dried with a rough towel, dressed and given breakfast. It may be preceded by shampooing for 5 or 10 minutes. Many young children cannot stand such stimulating and vigorous treatment, remaining chilly and blue afterwards, getting no healthy reaction. Such children should be washed in warm water, and, while sitting in it, have a cold douche at about 70° F. applied with a large sponge or from a jug to the shoulders and spine, followed by immediate



drying. After the age of 4 years the child, if strong and vigorous, may have a cold plunge bath, a cold sponge bath, a spinal douche, or a hot bath followed by a cold douche or shower. In addition, children should have a hot bath once or twice a week, ten minutes' duration, at bedtime, to thoroughly cleanse the skin. A daily cold bath is insufficient.

#### *The Temperature of the Bath.*

*Hot Bath.*—98-100° F., or 95° F. and raised to 105° F., and in some circumstances up to 110° F.

*Warm Bath.*—90-98° F.; usually 90° F.

*Tepid Bath.*—70-90° F.; usually 80° F.

*Cold Bath.*—60-70° F.

The *Hot Bath* is not contra-indicated by fever. It should last from 3-5 minutes, if at a high temperature, 5-15 minutes at the ordinary temperature, and be followed by a blanket and hot bottle. It is stimulant, useful for collapse, and induces sweating. *Hot packs* are sometimes more readily applicable. A towel or sheet is wrung out in hot water and wrapped round the child; then a blanket is wrapped round, a hot drink given, and the child left in the pack for 15-20 minutes. A *hot compress* or *fomentation*, changed every 10 minutes, is useful for raising the temperature in babies. *Vapour baths* and *hot-air baths* are sometimes needed. A woollen blanket is put under the child and the nightdress kept on. The bed-clothes are raised above the patient by a wire screen, and the air can be heated by an arrangement of electric lights, or moist air provided from a steam kettle, or by wrapping stone bottles, filled with boiling water, in very wet towels and then in flannel.

Hot packs and hot bottles, applied for too long a time, may cause hyperpyrexia. Baths can be made more stimulating by the addition of sea salt, 1-3 per cent. Sea baths have a similar effect. *Carbonated salt baths* are used in cardiac dilatation, and can be prepared in the home from the Zana Carbonated Effervescent Bath Powders. Salt must not be used unless the skin is healthy.

*Sitz baths*, at 85-95° F., for 3-10 minutes, are of value in pelvic affections, and at the onset of the catamenia. *Cold water* is used for therapeutical purposes in a variety of ways. The *cold bath* or *pack*, and modifications thereof, are of value for the reduction of high temperature, restlessness and delirium, the heat of the body being withdrawn by conduction. The child should sit in water at 90-95° F. up to the level of the lower ribs. The water is applied gently to the chest and head, and then the child is immersed and the temperature of the bath is cooled down to 60-75° F. by adding cold water or ice. Keep the patient in for 10-30 minutes, or until the temperature, which should be taken every few minutes in the rectum, is reduced to 101-100° F. Then remove to a hot blanket with a hot bottle to the feet. If the child gets blue, faint, or



exhausted, remove from the bath, wrap up in hot blankets, and give stimulants. Strictly speaking, the above is a warm bath gradually cooled. It is not so efficacious, but is less alarming than the cold bath given at a temperature of about 70° F. For babies the temperature of the bath should be 95° F. at first, and can be reduced 10 degrees. The baby should be gently massaged, kept in 5-10 minutes, finally wrapped up in a hot blanket and given a hot bottle. It is advisable to give an initial dose of stimulant before a cold bath or pack. Keep the head cool by ice, towels wrung out in cold water, or Leiter's coil. Remember that the temperature continues to fall after removal from the bath or pack. If the child is alarmed, a large Turkish towel or an old blanket can be spread over the bath, the child placed on this and allowed to sink in gently.

The *cold pack* is less alarming than the bath, and often quite as efficacious. It can be applied in two ways:—(1) Wrap the child in a sheet or Turkish towel wrung out in cold water, 60-70° F., apply a cold compress or ice to the head, and cool the body still further by rubbing with ice, or cover with an old blanket and leave in for 5-15 minutes. The bed should be protected by a rubber sheet. (2) Wrap the child up in a sheet dipped in warm water, and then in another sheet wrung out in cold water. Apply cold water or ice, with gentle friction as needed, for 5-15 minutes; then dry briskly until the skin is glowing. The *brandy pack* is a modification of the cold pack, the sheet being wrung out in a mixture of 1 part of brandy or Eau de Cologne to 6-10 of water at 60° F., and the child is then wrapped up in the blanket and left in for 3 hours. In the *cross pack* a linen bandage is wrung out in cold water and applied in a figure of eight fashion to the back and front of the chest and across under the armpits. It is next covered in a like manner with a thick single flannel bandage, and is left on all night. On removal, rub the chest with a cold wet towel and dry thoroughly. It is unsuitable for infants. Cold is also applied in the form of *compresses* and *fomentations*. Several layers of gauze are wrung out in water at the room temperature, and applied to the throat, thorax, etc., and covered with rubber or oiled silk. Unless renewed every 15-30 minutes it becomes a hot compress. Or a thick Turkey towel is wrung out in tepid, cold, or iced water, and applied from the top of the chest to the pubes, covered with flannel, and changed every 10 minutes. This is useful when it is undesirable to move the patient. It may be applied to the abdomen only for 2 hours twice a day, being renewed every 15-30 minutes. *Ice bags* relieve local pain, and can be applied directly or on a cold compress. They have been strongly advocated in the treatment of pericarditis, pneumonia, and pleurisy, but I am not often in favour of them. If applied in pericarditis, see that the right auricle is not over-distended, and that the extremities are warm.

Leiter's tubes can be used for the application of either hot or cold water to the head and other portions of the body. The cold compress is



useful in many local affections for the reduction of local heat and inflammation, and must be kept cool. A hydropathic one is applied lukewarm, allowed to get warm and to produce local congestion. Cold is further applied by simply bathing with cold water, e.g., the throat, for colds, and the feet, for cold feet.

In the reduction of fever the first measure commonly adopted is sponging hourly with water at 60-90° F. for 5-10 minutes, and the loss of heat by evaporation is encouraged by adding brandy, Eau de Cologne, rectified spirit, or vinegar. The child should have a rubber sheet on the bed, be wrapped up in a blanket, and sponged limb by limb, not completely exposed at once. Subsequently it can be put to bed, covered with a sheet, and the bed-clothes raised over a wire screen, open at the bottom end to allow the free circulation of air.

Mustard is a powerful cutaneous stimulant in the form of a bath or pack, and is valuable for convulsions, prostration with cold extremities and pallor of the skin, and collapse. The *mustard bath* is prepared by adding a tablespoonful of ground black mustard to about 3 gallons of water at 85-105° F. The action of the mustard is reduced by temperatures over 85° F. In this the child, with a cold compress on the head, and well covered in at the neck, is immersed for 5-15 minutes, and then wrapped up in hot blankets. The strength of the bath should be such that the forearm of the nurse can be kept in for a minute without more than tingling. Stronger baths are made by adding 1-4 oz. to each gallon. The mustard can be made into a paste with cold water, and stirred in gradually or suspended in a linen bag in the water. A *mustard pack* consists of a sheet, towel, or several folds of muslin, wrung out in a quart of tepid water, to which a tablespoonful of mustard has been added. This is wrapped round the thorax or body, covered with a blanket, and left on for 5-15 minutes, or until the skin is reddened. If applied to the chest, a cotton wool jacket should be put on afterwards. A stronger pack is made by wringing out the towel in a liquid paste of 1 lb. of mustard to 3 pints of cold water. It is applied for 5-15 minutes, and the child then washed in warm water and wrapped in hot blankets.

A large variety of medicinal baths are available, chiefly for skin affections. The administration of drugs by this means is uncertain in degree and unsatisfactory, although absorption is rather more marked in the young. We may classify these baths :—

*Palliative*.—Bran, gelatine, or starch (1 oz. to 2 gallons), milk and water; useful for itching affections.

*Alkaline*.—Borax, carbonate of soda, soft soap (1 oz. to 30 gallons); if the skin is irritated and thickened.

*Astringent*.—Decoction of oak bark; for moist eruptions.

*Disinfectant*.—Permanganate of potash.



*Antiparasitic.*—Potassium sulphide or sulphurata (1-4 oz. to 30 gallons); or sulphaquea if the bath is a metal one. It can be used also in dry eczematous conditions.

*Tar Bath.*—Add oil of cade or liquid carbonis detergens (from a teaspoonful to 2 tablespoonfuls per gallon).

*Mud and Peat* baths are recommended for affections of the lymphatic system, and are best carried out at health resorts.

*Spa treatment* generally consists of salt and brine baths, and is not as useful or available for children as for adults.

*Sea bathing* can be begun after the sixth year. The best time is in the morning before breakfast, after a glass of hot milk and a biscuit, or 2-3 hours after a meal, but not while chilly or sweating. The head should be immediately wetted. Duration 15-20 minutes. It should be followed by food and exercise. Otorrhœa is a contra-indication.

*Physical methods* include simple rubbing of the skin, massage, abdominal massage, passive and resisted movements, and muscular exercise in bed. These are only applicable, with certain exceptions, after early childhood. After the fourth year we can make use of games, exercises and gymnastics, drill, fencing, running, rowing, and swimming. School life should begin in the eighth year. Summer camps deserve more cultivation than they receive in this country. Rest cures are occasionally needed.

**Local Applications of Heat** are generally made by means of hot bottles, preferably indiarubber ones, bran bags, fomentations, flannel or spongiopiline wrung out in hot water, changed frequently; or by poultices of bread, linseed, starch, etc. They cause an active local hyperæmia, increased local leucocytosis, and the rapid removal of deleterious products or pus formation. Applied to an acutely inflamed surface, or an unbroken boil, a poultice affords very considerable relief, besides protecting it from injury and infection. In its action it is comparable with that of Bier's cupping apparatus, and leads to cure by local auto-inoculation. Its mental effect on the patient and friends is considerable, and undoubtedly it often affords relief. Although somewhat messy, it is easier to manage than a series of hot fomentations, wets the bed less, and retains its heat and moisture longer, especially if covered with guttapercha tissue. Certain varieties are worthy of mention.

The *sugar poultice* or fomentation consists of a hot 10 per cent. solution of cane sugar, to which 1 per cent. of citrate of potash is added. Lint or flannel is wrung out in the mixture, applied every 4 hours, and covered with guttapercha and wool. It is cheap, handy, and painless. The sugar acts as an osmotic, attracting the lymph to the surface, and the citrate prevents local coagulation on the surface. Such a poultice is very useful for boils and carbuncles, after incision. Creolin or boric acid can be added.



**Starch Poultice.**—Mix a teaspoonful of boric acid and a tablespoonful of best white starch with cold water, and then add a pint of boiling water, stirring well. A thick mucilaginous mass is formed. Spread the jelly, when cold, thickly on cotton, and cover it with a piece of muslin. Put it on at night and remove next morning. It is very useful for cleaning the skin, especially in affections of the scalp.

Local applications of heat, applied to the feet and back of the head, relieve headache. They are valuable in colic, toothache, neuralgia, croup, sore throat, rheumatism, pleurisy, peritonitis, congestion of the lungs, and other affections. In applying a poultice to babies it should be large enough for the infant to lie in, and not extend on to the front of the chest.

**Counter-Irritation.**—The severe methods of blistering and vesication, of escharotics to create sloughs or ulcers, and setons are almost never required. Blistering with cantharides is liable to set up nephritis. Methyl iodide, sprinkled on bibulous paper and applied, is stated by Causson (1906) to be cleaner and more powerful than cantharides, and not to cause nephritis. Usually it is quite enough to use simple measures, enough to produce rubefaction of the skin, and even they must be used with caution, for the skin is very delicate and the nervous system very sensitive to impressions. The local effects are dilatation of the blood vessels, stimulation of the sensory nerve endings and pain, and reflex stimulation of the vaso-motor system and consequent contraction.

Electra cloth, impregnated with Chillie paste, and the mustard leaf poultice, pack, or bath are the best counter-irritants. The leaf is simple, clean, easily applied, and easily controlled in its effects. It can be cut to the required shape, requires no special preparation, and can be applied in almost any place and for any length of time. The poultice is made of 1 part of mustard and 8 of linseed or flour. The mustard is rubbed up in cold water and well mixed with the boiled meal. A temperature above 85° F. or so weakens the action of the mustard. Put on a pad of cotton wool afterwards. The pack and bath are described under the section on Hydrotherapy. Counter-irritation can be applied by rubbing in camphorated oil or turpentine liniment, or by turpentine stupes, a towel wrung out in a quart of hot water to which turpentine (1-8 drs.) has been added.

**Dry Cupping** is useful in nephritis and congestion of the bases of the lungs. A modified form is much used nowadays in the treatment of various diseases, under the name of “passive hyperæmia” induced by Bier’s suction cups. These are glass cups attached to rubber balls, whereby suction can be exerted. The method is useful for the production of local congestion and auto-inoculation in the treatment of boils, carbuncles, enlarged glands, sinuses, and tuberculous affections.

**Bleeding.**—Infants do not stand the loss of blood well, and older children not so well as adults. Local bleeding, by one or more leeches, relieves



the pain of acute otitis, the headache of meningitis and the pain of pleurisy. Applied below the right nipple the loss of blood is beneficial in the cardiac dilatation of bronchitis, pneumonia, acute pericarditis and myocarditis, and mitral stenosis. A leech to the septum nasi may possibly arrest the onset of fits due to cerebral congestion. Bleeding is also useful in uræmic fits and infantile eclampsia. Two leeches are sufficient for an infant and 3-6 for an older child. General bleeding, with the abstraction of not more than 3 or 4 oz. of blood, is sometimes judicious in the severe cyanosis of pneumonia from dilatation of the right heart. Wet cupping must be avoided.

**Pharmacotherapy.**—In estimating the value of treatment it must be remembered that tissue metabolism is much more active in children than in adults. A judicious scepticism is imperative or undue importance will be ascribed to drugs. No medicine should be given without a definite object. If a placebo is given, let it be palatable. Never give an unpalatable medicine if it can be replaced by one pleasant to take. Give drugs in small, frequent doses, rather than in large, occasional ones. Disguise the taste of nauseous ones as far as possible, and give all medicine in such a form as to be swallowed easily. Fluids are taken better than pills or powders. The dose should be 1 teaspoonful for infants and 2 for older children. A measure glass should always be used for domestic spoons vary greatly in size. In infants it is of the greatest importance to avoid drugs liable to cause anorexia, nausea, or gastric disturbance. Flatulent distension of the stomach and bowels is a serious complication, for instance, in pulmonary troubles, and is liable to be set up by rash medication. Choose by preference simple solutions, concentrated tinctures, fluid extracts, alkaloids and their salts. Bitters are generally disliked, unless well diluted, and are not often needed. Agreeable preparations are syrups, chocellæ, and tablets or tabloids, dissolved in water for infants.

Take into consideration the child's age and mental calibre, the character of the parents, the dose required, the relationship to meals and sleep, and the frequency of administration. For instance, phosphorus is given once daily, opiates according to their effect, and salicylates in full and frequent doses. Drugs, such as chloral, bromides, and mercury, are well borne by infants, but opiates must be used with great caution. Belladonna, digitalis, and quinine are also well borne by the young. The dose may be estimated in accordance with the age and size of the child. If by weight, the dose for an infant is one-fifteenth that for an adult. If by age, take the age of the child at its next birthday as the numerator and the age *plus* 12 as the denominator of the fraction necessary. Thus, if the adult dose is 1 gr., the dose for a child in its third year is  $\frac{3}{12+3}$  or  $\frac{1}{5}$  gr. Another method is to divide the age of the child at its next birthday by 24, giving a child of 3 years a dose of  $\frac{1}{8}$  gr. Under



one year of age let the dose be equivalent to one-twelfth of that suitable at one year for each month of life.

The chief *cathartics* are castor oil, calomel, sodium and magnesium sulphate, sodium phosphate, citrate of magnesia, calcined magnesia, milk of magnesia, rhubarb, manna, cascara, senna, and aloes. Epsom salts may be given in the form of a saturated solution, with the addition of 1 dr. of aromatic sulphuric acid to 4 ozs., in doses of 1 or 2 drs. with a little water.

*Diuretics*.—Acetate of potash, liquor ammon. acetat. (Mindererus spirit), bitartrate of potash (cream of tartar) grs. 1-10. Imperial drink: 1 dr. of cream of tartar added to boiling water and a little lemon juice, given cold. Diuretin.

*Diaphoretics*.—The spirit of nitrous ether, pilocarpin.

*Febrifuge*, and as a pleasant drink in stomachic disorders.—Use an effervescing mixture made by mixing in equal quantities citric acid 3 drs. to water 6 oz., and bicarbonate of potash 2 drs. to water 6 oz., the alkali not being completely neutralised.

*Antacids*.—Bicarbonate of soda, sal volatile, milk of magnesia.

*Stomachics and Carminatives*.—Aromatic water of dill, caraway, cinnamon, aniseed, and peppermint.

*Expectorants*.—Wine of ipecacuanha and antimony, chloride of ammonia, liquorice, tolu, senega, squills. The addition of bicarbonate of soda, liquor potassæ, or iodide of potash, helps by making the secretions more liquid.

*Pulmonary Sedatives and Disinfectants*.—Heroin gr.  $\frac{1}{200}$  to  $\frac{1}{24}$  terpene hydrate, creosote and carbonate of creosote (creosotal), guaiacol and its carbonate (duotal).

*Intestinal Astringents*.—Chalk mixture; carbonate, subnitrate and salicylate of bismuth; kino, catechu and tannin rarely. Turpentine in the form of emulsion, e.g. Ol. Terebinth. drs. iii, Pulv. Acaciæ  $\frac{1}{2}$  oz., Sacch. alb.  $\frac{1}{2}$  oz., Sp. Lavandulæ co. drs. iii, Aquæ ad 6 oz. Dose, dr. i-ii t.d.s.

*Intestinal Antiseptics* are considered under the treatment of diarrhœal affections and *Anthelmintics* in the chapter on worms.

*Urinary Antiseptics*.—Urotropin, well diluted with water; salol and benzoate of ammonia. Large doses of citrate of potash render the urine alkaline and do good in colon infections of the bladder, for the bacillus will not grow in an alkaline medium. Acid sodium phosphate.

*Antispasmodics*.—Bromides and various combinations thereof, bromoform, pertussin (an extract of thyme), belladonna and its alkaloids.

*Alkalies* are valuable in moderate doses in all catarrhal and ulcerative affections of the mucous membranes, and as a local application. The sodium compounds are less depressant than the potassium salts. They reduce the acidity of the blood and urine. When given before meals they



dissolve mucus, and in efficient doses encourage the secretion of acid. After meals they may be given to reduce hyperacidity. The liquor potassæ or liquor sodæ, and other alkalies, are useful in acute bronchitis with viscid secretion. Sodium citrate added to milk prevents curdling in the stomach. The bicarbonate is of value as a local application in eczema, whitlows, after incision, ulcers (grs. 15 ad 1 oz.) and in toothache.

*Salicyl* compounds are used in rheumatic affections, acute colds, many febrile conditions, and as diaphoretics. Salicylate of soda, salicin, salicylic acid and aspirin (acetyl-salicylic acid) are the best preparations. Aspirin cannot be combined with an alkali. It has a more prolonged action than the other compounds, for it is not nearly as rapidly excreted. Many of these compounds are only active because they are converted into salicylic acid in the body. Salicin, aspirin and methyl-salicylate, applied locally, act in this way. Benzoic acid is a valuable anti-rheumatic remedy, but inferior to salicylic acid. Large doses of these drugs can be given if the bowels are kept open, and if alkalies are added in large amounts. Further consideration of them will be found in the chapter on acute rheumatism.

*Quinine* can be given in warm chocolate, shaken up with syrup, mixed with glycerine, 1 gr. to the drachm, and given in milk; in gelatine capsules; mixed with scraped raw apple. An effervescing mixture is made by adding sulphate of quinine gr. i, citric acid grs. iii, and syrup of orange 1 dr., to a solution of bicarbonate of soda in water. Or it may be given as a tannate in powder or chocolate tabloids, or as euquinine. It can be rubbed into the skin, 1 dr. to the ounce of benzoated lard; given in suppository; as an enema; or subcutaneously, one part of the hydrochlorate in four of water.

*Iodine* is rather difficult to administer. Wingrave advises that 5 gms. of iodine should be dissolved in 76 c.c. of 90 per cent. alcohol, and added to 8 gms. of tannic acid and 60 c.c. syrup. It is heated without boiling for 20 minutes, and more syrup added up to a total of 150 c.c. The dose is from  $\frac{1}{2}$ -2 teaspoonfuls in water or wine before meals. It contains 2 grs. of iodine in each drachm, and is somewhat similar to "Vin Nourry." Smaller doses should be given to children. Tincture of iodine in drop doses is given to stop vomiting. Iodine ointment contains iodine gr. i, pot. iod. dr. i, vaseline oz. i. It can be made with glycerine, instead of vaseline, for application to mucous surfaces. The iodides of sodium and potassium are of great value in many diseases.

*Creosote* is used as an intestinal disinfectant, anti-fermentative, and in pulmonary affections. One part of best creosote, with one part of a 50 per cent. solution of pure sodium salicylate, and two parts of concentrated decoction of quillaia (1 in 7), forms a clear solution. Or it can be given in capsules. In mixture the taste can be disguised by spirits of chloroform, ext. glycyrrhizæ liq., and inf. gentian. co.; by tinct. gentian., rectified spirits, and sherry wine; or by syr. tolu and rum. As an ointment



it is prescribed with equal parts of lard, olive oil, and lanolin,  $\frac{1}{2}$  dr. to the ounce.

*Mercury* is generally given in the form of calomel, grey powder, or blue ointment. The immediate resort to grey powder in every infantile ailment is by no means to be recommended, but should not be spoken of contemptuously. Many a time when the so-called inevitable dose of grey powder has been omitted, it has been to the child's disadvantage. Its use in syphilis is too well known to require comment further than in the section on this disease.

*Cod-liver oil* is the best of tonics, and a food also. It can be given in innumerable ways and combinations, as with maltine, syr. ferri. phosph. co., vinum ferri, glycerin, syrup of orange, lime water, and caraway water ; or as :—

Tinct. calumbæ m. v, ol. mor. m. xxx, aq. carui ad dr. i ;

Ol. mor., syr. calcis lactophosph., aq. calcis aa m. xviii, sod. hypophosph. gr. i, mucil, acaciæ m. v, ol. cassiæ m.  $\frac{1}{4}$  ;

Ol. mor. oz. ii, maltine oz.  $\frac{1}{2}$ , syr. calc. hypophosph. oz.  $\frac{1}{2}$ , glycerine drs. ii, pulv. acaciæ drs. ii, aq. cinnamomi ad 4 oz.

*Iron* is given in the form of organic compounds, as in yolk of egg, meat juice, potatoes, spinach and other green vegetables, and various preparations from blood, such as hæmatogen. The inorganic salts, least likely to upset the digestion, are the citrate of iron and ammonia, tartrate of iron, dialysed iron, reduced iron, and the saccharated carbonate of iron, in doses of  $\frac{1}{2}$ -5 grs. The reduced iron does not discolour the teeth, and can be given in sandwiches. A good mixture for simple anæmia and debility contains the citrate or tartrate grs. i-iii, pot. cit. grs. v, sp. amm. arom. m. v-x, glycerine and inf. calumbæ. It is beneficial in chronic gastric catarrh, an hour before meals, after a course of alkalies. The acid salts, such as the perchloride and the sulphate, are more powerful, and useful for tuberculous affections, rickets, splenic anæmia, purpura, and other blood dyscrasia. For enlarged glands a useful mixture contains the tincture of the perchloride of iron m. v-x, liq. hydrarg. perchlor. m. v-xv, liq. arsenici hydrochlor. m. ii-v, made up with syrup of orange and chloroform water. The perchloride can be pleasantly given in aerated water after food. *Iron Lemonade* is made of tinct. ferri perchlor. drs. ii, acidi phosph. dil. oz.  $\frac{1}{2}$ , syr. limonis oz.  $\frac{1}{2}$ , syrupi ad oz. vi. Dose : 1 dessertspoonful in water after meals. A good mixture for the convalescent stage of acute parenchymatous nephritis contains the tincture of the perchloride m. v, liq. ammon. acetat, m. xv, ac. aceti dil. m. ii, water ad drs. ii, given after food in aerated water. The sulphate can be combined in mixtures with the sulphate of magnesia. During a course of iron a saline aperient should be given twice a week before breakfast. Other compounds often used are the syrup of the phosphate, the compound syrup of the phosphate (Parrish's or Squire's Chemical Food), and the syrup of the iodide, especially



useful in tuberculous affections and the resolving stages of chronic lung affections.

*Arsenic* is a valuable gastric sedative. Minim doses of Fowler's solution often stop reflex vomiting, colic, or diarrhœa. In doses of 3-5 minims it is a valuable hæmatinic. In large doses it is given for chorea (q.v.), but may give rise to peripheral neuritis, skin pigmentation, keratosis, etc. It is generally contra-indicated in acute skin affections and valuable in chronic ones. The liq. arsenici hydrochlor. must be used in acid mixtures. The stronger preparations of arsenious acid are not often used for children, e.g. the acid, the iodide, and soamin or atoxyl.

*Coal Tar Products.*—Phenazone (antipyrin) is used for the reduction of temperature, the relief of nervous symptoms, headache, convulsions, enuresis, etc. Phenacetin is less dangerous, and almost, if not quite, as efficacious, especially for the relief of headache. Still, young children stand phenazone very well. Antefebrin is contra-indicated, for it is unnecessarily powerful and dangerous. These drugs are valuable nerve sedatives.

Phenacetin is not soluble, so is given in powders. The combination of 1 gr. with  $\frac{1}{4}$  gr. of caffein citrate is very valuable. Antipyrin is soluble, but does not taste nice. One grain doses in a drachm of syrup of orange can be given hourly at one year of age. The addition of  $\frac{1}{2}$  gr. of codeia to 2 oz. of the mixture makes it more efficacious as a sedative and antispasmodic. As antipyretics these drugs must be used with caution, and should not be given unless the temperature is persistently at or over 104° F. They produce their results by acting on the nervous and muscular mechanisms of the circulation and respiration, and are powerful depressants. It may be necessary to give them for the reduction of high fever when the attendants are stupid or ignorant, and efficient nursing is not available. In the hyperpyrexia of acute rheumatism, in enteric fever, and bronchopneumonia they are sometimes permissible; still more rarely in diphtheria, specific fevers, and croupous pneumonia. In all these affections the myocardial degeneration or inflammation, so apt to occur, makes them dangerous remedies. On the other hand, frequent small doses of either antipyrin or phenacetin are rarely injurious, lessen the frequency of the pulse, and often increase sweating. The use of synthetic drugs in fever is based on the false idea that the malaise is due to the fever. Large doses for infants are unnecessary and often dangerous. The smaller doses recommended act more as nerve sedatives and are harmless. Any depressant effect can be counteracted by a few drops of brandy.

*Bromides* can be given freely at all ages, and act simply as nerve sedatives. Up to 4 grs. for a dose every 2 hours may be given even in the first month of life, but such large doses are rarely needed. Bromides are useful in all forms of nervous irritability, convulsions, reflex colic, and some cases of enuresis. Bromural, a combination of bromine, valerian,



and urea, is a harmless and mild hypnotic in doses of 5 grs. Occasionally a child shows a marked susceptibility to bromides, and develops a rash, which may be extremely severe and disfiguring, even though only a few grains have been given. The liability to acne spots of a mild type can be counteracted by adding a few minims of liq. arsenicalis to the mixture.

*Chloral* is without doubt the most valuable drug for all kinds of infantile convulsions. During the first year it may be given in doses of 1 gr. for each 3 months of life, combined, if desired, with double the amount of sodium bromide, and given in syrup of orange. Under supervision, reducing the amount according to the effect, it may be given every 2 hours, and in even larger doses. If it cannot be swallowed, it can be given by rectal injection in doses of 5-10 grs. every 6 hours, dissolved in water. This method is particularly valuable for a child actually in convulsions. Frequent small doses are useful in severe chorea, but large doses seem to be sometimes dangerous. The syrup contains 10 grs. in each drachm, and can be given in soda water or lemonade.

*Hypnotics.*—Bromural, bromides, and chloral meet the needs of most children. Phenacetin and antipyrin suit others. Veronal, trional, and sulphonal are rarely necessary.

*Opium* and its preparations are occasionally absolutely necessary. They must be used with great caution, not indiscriminately. Codeia is often preferable to morphine. These drugs are specially valuable for the relief of pain and laryngeal spasm, the prevention of peristalsis in diarrhoea after free evacuation of the bowels, and as hypnotics. Small doses must be given during the first year of life. The dose of codeia is gr.  $\frac{1}{300}$ , morphine  $\frac{1}{1000}$ , Dover's powder gr.  $\frac{1}{20}$ , tincture of opium m.  $\frac{1}{20}$ , paregoric m. i., per month of life in the first year, and can be given every 3 or 4 hours.

*Stimulants.*—The most powerful cardiac stimulants are given subcutaneously, e.g. strychnine gr.  $\frac{1}{400}$  -  $\frac{1}{60}$ ; Merck's digitalin gr.  $\frac{1}{400}$  -  $\frac{1}{20}$ ; a 10 per cent. solution of camphor in sterilised oil m.  $\frac{1}{6}$ -3.

*Alcohol* is a useful and necessary medicine for infants and children, but is often given too readily and in too large quantities. It must be looked upon as a powerful drug, and only ordered when necessary. It should be given well diluted, in small and frequent doses, e.g., 3-10 minims every 2-3 hours in the first year of life, and not continued for long after recovery. It is used to tide a child over a crisis in an acute illness, as a soporific in fever, and as a gastric stimulant in chronic ailments. It is beneficial in collapse, in septic conditions of all kinds, and in the cardiac asthenia of diphtheria, pneumonia, typhoid fever, scarlatina, and measles. Generally speaking, it is contra-indicated in cerebral and bronchial affections. Epilepsy, chorea, and neurasthenia have been ascribed to the too free use of alcohol in early life, and the frequent stimulation of the gastric mucosa is said to induce dyspepsia, gastritis, and even hepatic



cirrhosis. Certainly, acute gastritis in a baby has, in my experience, been due to champagne, but this was not the result of medical advice. Brandy is the most reliable form, diluted with water or added to the medicine. Other spirits may be used. Pure rectified spirit never seems to produce as good results, probably because of its lack of ethers. Champagne, iced in cases of vomiting, good hock, and white wine whey are useful. A child generally prefers the non-sparkling wines. Burgundy, port wine, light ale, and stout are valuable in convalescence. Malt, in the form of Guinness's stout, mixed with cream and sugar, is valuable in infantile malnutrition. Another good food is brandy and egg, made of the yolk of one egg, sugar dr. i, brandy m x, cinnamon water dr. i.

**Cutaneous Therapy.**—Simple massage of the skin is useful to improve the circulation and nutrition. Precede it by a bath at 90-100° F. for 10 minutes. Dry by gentle rubbing with a hot rough towel, and then rub with warm olive or cod-liver oil or benzoated lard, and put on a flannel nightdress. Drugs may be given in this way, but the amount of absorption, viâ the gland ducts, is uncertain. The oleate of mercury and blue ointment are commonly applied by this method, a lump the size of a large pea being rubbed into the skin of the abdomen and covered with a flannel binder. The red iodide of mercury ointment is used for glandular enlargements. Some drugs are readily absorbed by the skin, notably carbolic acid, which is consequently a dangerous drug in early life, and salicylic acid and its congeners, especially methyl salicylate and betul oil, a preparation containing this compound. Quinine and digitalis can be absorbed in small quantities.

Endermatics are rarely used. The skin is removed by blistering, painting with pure carbolic acid, or moistening a watch glass with strong ammonia and inverting it over the skin.

*Hypodermic medication* should not be used for children under 3 years of age, except after great experience and in serious collapse. Dangers arise from idiosyncrasy. It is not advisable for older children, because of the pain and fright, and rarely needful for the relief of pain. If necessary, the skin can be numbed by ethyl chloride spray, ether, or rubbing with a menthol cone. The antitoxin for diphtheria is the drug most often given in this manner. Morphia, gr.  $\frac{1}{100}$  under 1 year of age, may be required in otitis media, renal colic, uræmia, and the status epilepticus. Pilocarpin is sometimes given in daily or weekly doses to remove exudations, and to induce sweating in uræmia. It should be preceded by a warm bath and blanket wrap, and given at night. After the sweating is over put on a warm nightdress. Give it in small doses tentatively, for it is quite impossible to estimate individual susceptibility. Atropin, in doses of  $\frac{1}{400}$  -  $\frac{1}{200}$  of a grain, counteracts the bad effects, namely, weak pulse, vomiting, coldness, depression and threatened collapse. Liquorstrychninæ, ether, brandy, musk, and caffein are given for cardiac failure. Ether is



liable to cause sloughing of the skin. Adrenalin solution is of little or no value.

*Transfusion*, i.e., subcutaneous injection of saline fluids, is used for collapse, shock, loss of blood after operations and in hæmophilia etc., severe vomiting and diarrhœa, to promote elimination in septic states, and in coal gas poisoning. Intravenous injections have no additional advantage, and are not necessary. The chief effects are a rise of blood pressure and stimulation of the kidneys.

The apparatus consists of a glass cylinder, such as that of a syringe, 2-4 feet of rubber tubing, and a Dieulafoy's or long hypodermic needle, all of which are carefully sterilised. Disinfect the skin at the site of injection. Insert the needle for about 2 inches under the skin of the abdomen, into the side of the thorax directed upwards towards the axilla, below the clavicles, or on the inner side of the thighs. A pressure of 18 inches is sufficient when once the flow has started. Inject 3-4 oz., or less in infants. It will flow in through one puncture in about 15-60 minutes. Close the puncture with cotton and collodion, and massage gently. The rate of absorption varies. The only complications are subcutaneous hæmorrhage and abscess formation. Use for injection normal saline 0·9 per cent. strength; as a stimulant, sod. chlorid. 2·5, caffein. citrat. 0·75, sterile water 300 c.c., in doses of 1-4 drachms every 4 hours; or a mixture of sod. chlorid. grs. 180, potass. chlorid. grs. 12, sod. sulphat. grs. 100, sod. carbonat. grs. 100, sod. phosphat. grs. 8, in 8 oz. of distilled water, made up to 2 pints for use. These fluids should be sterilised by boiling for 10 minutes, and at a temperature of 120° F. when poured into the cylinder.

The subcutaneous injection of nutritive materials, such as 6 per cent. dextrose solution and yolk of egg, is occasionally employed.

**Rectal Therapy.**—The rectum is used as a channel for the administration of water and food for the maintenance of life, in the reduction of the body temperature, in the treatment of collapse, in the treatment of local and general diseases, such as colic and tenesmus, abdominal distension, colitis and other diarrhœal affections, hæmorrhage, prolapse of the rectum, and threadworms; and for the introduction of drugs which, for various reasons, cannot be given by mouth or subcutaneously. Rectal absorption includes absorption by the colon, for solids and fluids pass upwards into the colon as far as the cæcum, and even further, in consequence of reflex peristalsis. Insoluble substances may reach the stomach. Cases in which rectal feeding has proved unusually satisfactory are probably those in which the reflex peristalsis is considerable. The capacity for absorption varies with the individual and the nature, rather than the percentage composition, of the nutrient fluid. Irrigation of the colon is necessary for cleansing, previous to giving a rectal feed, to empty the bowel and wash away the decomposing material left from previous feeds.



It should be done once in 24 hours. Irrigation is also valuable in the relief of thirst, the reduction of fever, to counteract collapse, and to wash away mucus, toxins, bacteria, and degenerated epithelium. It must be carried out by a doctor or skilled nurse. An inexperienced person is liable to cause damage or perforation of the gut. Use salt solution, 1 teaspoonful to the pint of water.

Place the child on the left side with the hips raised on a pillow, on the back with the buttocks raised, or in the genupectoral posture. The best position for a baby is on the back or the attendant's lap, with the buttocks raised. A rubber sheet must be so arranged that the escaping fluid flows into a basin or other receptacle. Use a soft rubber catheter, No. 9-12, attached to a rubber tube 3 or 4 feet long, with a glass funnel inserted at the free end. Oil the catheter well and pass it upwards slowly and gently as the fluid flows in and distends the bowel, opening out the folds and enabling the tube to be pushed steadily higher. It is infinitely easier to get good results when the fluid is injected into the colon. If it only distends the rectum it will be evacuated suddenly and forcibly. Only use moderate pressure, holding the funnel at a height of one to two feet above the level of the anus. Gentle manipulation of the abdomen assists the flow up the colon. Normally some of the fluid escapes by the side of the catheter from time to time. When a sufficiency has been injected, compress the buttocks with the hand and keep the fluid in for a time. Then detach the tube and allow the fluid to flow out through the catheter. For cleansing purposes the water should be lukewarm, about 80° F. The colon will hold one pint at six months and double that amount at two or more years of age. The injection may be repeated several times if the child is not distressed.

**Rectal Feeding** is an overrated means of nutrition. Nutrient suppositories are useless, and nutrient injections not much better. What little benefit there is, depends on the fluid rather than the nutritive constituents. Simple saline injections are generally as beneficial. Even as a means of giving the stomach a complete rest the method is unreliable, for the introduction of nutrient material into the lower bowel will cause gastric secretion, pain, and vomiting. In the colon cane sugar can be converted into grape sugar, and a certain amount of starch into dextrin. The continuous or drop method is unavailable, unless the child is comatose. Otherwise it will not keep still enough. For injection we may use plain water, normal saline fluid, dextrose 6 per cent. solution, cane sugar, dextrin, and peptones. Peptones are liable to set up diarrhoea. Dried casein, e.g., protein, plasmon, and sanatogen, are little absorbable, and even egg albumin is not much better in this respect. Fat in the yolk of egg can be fairly well absorbed, and emulsified olive oil and milk fat to a less extent. Unpeptonised milk, starch and wines must be avoided. Brandy can be added. Salt is a useful addition to most mixtures.



The following feeds may be tried: (1) One egg-white, salt grs. x, weak starch solution, 3 oz. (2) Horse serum, with 5 grs. of salt per ounce. (3) Yolk of one egg, dextrose drs. ii, salt grs. x, water or peptonised milk to 5 oz. (4) Two drachms each of somatose, sanatogen, or plasmon, and of dextrose or dextrin, salt grs. x, and water or peptonised milk to 5 oz. This may be enriched by adding the yolk of an egg, and 10-30 drops of brandy. Dilute it, if too thick or if more fluid is needed. Even under the most favourable conditions the method is only a means of delaying starvation. From 1-2 oz. for an infant, and 2-4 oz. for an older child may be injected every 4 hours at first, and then every 6 hours.

The *reduction of fever* is brought about by irrigation of the colon with a gallon of water at 95° F.; by enemata of water at 70-80° F.; by the injection of small quantities of cold or iced water; or by ice suppositories. It is a useful means, if there is not much prostration, in cases of high fever in infants and young children.

*Collapse* is treated by injecting water or normal saline at 105-110°F. The addition of  $\frac{1}{2}$ -1 dr. of tincture of musk or of brandy makes the injection more efficacious. One to three minims of tincture of opium may be added for collapse due to severe pain.

*Pain* and *tenesmus* are relieved by iced water, hot water, starch solution with a few minims of tincture of opium, or cocaine suppositories gr.  $\frac{1}{4}$ , glycerine enema and hot applications to the abdomen are generally sufficient to relieve colic.

*Abdominal distension* is relieved and *flatulence* is evacuated by passing a rectal tube; by an enema of dill water, alone or with an equal quantity of chloroform water; or of  $\frac{1}{2}$ -2 drs. of tincture of assafoetida in water or starch mucilage. The treatment of constipation is discussed under that head.

For *irrigation* use simple solutions of water, common salt, or bicarbonate of soda, a drachm to the pint. As antiseptics add to each pint a drachm of boric acid, sod. salicyl., or sod. benzoat; naphthol 3-5 grains; thymol 5-10 grains. For astringents make use of 1 per cent. tannin lotion; tannic acid  $\frac{1}{2}$  dr. or ext. hamamelis 1 dr. to the pint, or to 3 or 4 oz. if it is to be retained; argyrol grs. 1-3, or nitrate of silver gr.  $\frac{1}{4}$ -1 in each ounce. Silver nitrate is the best astringent in chronic ulcerative colitis, more valuable than organic silver compounds. It is preceded, 3 hours beforehand, by an enema of salt and bicarbonate of soda. From  $\frac{1}{4}$ -1 pint of the silver solution is injected. If it causes much pain, give a small dose of morphia or laudanum. The injection may give rise to pain and vomiting, but never does harm, though the symptoms may be alarming. Constipation often follows, and must be relieved by a simple enema in 3 days time. Not more than 3 or 4 of these injections should be given, at intervals of 3 or 4 days,



unless definitely good results are obtained. Sedative injections consist of decoctions of gum arabic, starch, linseed, and various cereals.

*Drugs*, such as antipyrin, creosote, potassium iodide, quinine, etc., can be given in suppositories, but the method is unsatisfactory, as it is impossible to gauge the rate or amount of absorption. Chloral hydrate in watery solution is absorbed very readily.

*Objections* to the use of enemata and irrigations:—Mechanical injuries are not unknown. The continued use of enemata and suppositories is liable to set up catarrh of the mucous membrane. The effect on constipation is lost after a time, unless changes in the kind of enema are made. The enema rash is an important effect. It appears most commonly in young patients, between 6 and 12 years of age, after an enema for constipation, as a preliminary to operation, or for the cure of thread worms. It is not due to soap, for it may follow the injection of plain water. Probably, it is caused by the absorption of dissolved fæcal material. The rash is symmetrical; consisting of slightly raised patches of erythema which run into blotches, confluent like that of scarlatina, sometimes urticarial. It appears in 3-48 hours; on the front of the knees, extensor surfaces of the elbows and the buttocks; spreads to the cheeks and chin, the outer sides of the thighs, but not to the palms or soles. It may come out first on the buttocks, spreading upwards and downwards. It does not itch, and rarely desquamates. There is occasionally a little fever. The erythematous rash disappears in less than 24 hours, but the urticarial one may last 2 or 3 days. No treatment is needed.

**Fever, its Causation and Treatment.**—The body temperature of infants and children is very unstable. Fever is easily induced during the first 6 months of life, and up to puberty the temperature rises on slighter provocation than in later life. This is due to the rapidity of the circulation, oxidisation and growth, and the instability of the nervous system. Mere fright may raise a baby's temperature several degrees. A rise of temperature is due to increased production or diminished loss of heat, or a combination of the two factors. It may result from the application of external heat, e.g., by the continuous hot pack or by hot bottles, if the means of elimination are restricted. Protection from loss of heat is one cause of the maintenance of fever. It is imperative that for the reduction of fever the clothing should be light, and the room well ventilated. Pathologically a rise in temperature is ascribed to increased oxidation, set up by a combination of toxins with antibodies; varying with the virulence of the organism and the reaction of the host. Toxins produced in the tissues of the body or in the food, before or after ingestion, may directly affect the heat regulating nervous mechanism.

The temperature of the newborn babe is the same as, or slightly higher than, that of the mother, but soon falls to or below normal from exposure, bathing, and lack of food. It may reach the normal level in



24 hours, or not for 3 or 4 days, and then ranges from 98-99.4° F., being a fraction higher in the evening than in the morning. An occasional rise for a few hours has no special significance, but a daily rise, or the persistence of a temperature one degree above normal, requires investigation of its cause. A rise of temperature is indicative of mischief, but may occur without any evident cause or sign of illness. It is often overlooked, for the temperature is not systematically taken unless the child is ill. Half the cases are due to infection, one-fourth to gastro-enteric disturbance, a few to inflammation of the lungs and serous membranes, and in the residue the cause is doubtful.

*Inanition or Thirst Fever* is a name given to cases of fever during the first few days of life, on the assumption that sepsis rarely produces fever before the fifth day, and because the fever has subsided, sometimes quite abruptly, on the free administration of water or a plentiful supply of breast-milk. These infants show no sign of illness, though the fever may last for 3 or 4 days. Seeing that insufficient food generally causes fall in temperature, it is much more probable that the fever in these cases is due to mild sepsis, to the absorption of abnormal intestinal products, or to intestinal or renal irritation.

In investigating the sources of fever, examine for teething and local affections of the mouth, throat, and naso-pharynx. Sore throats are often overlooked, for a child may not complain of pain, while chronically enlarged and pitted tonsils may give rise to fever through the decomposition of food and secretions retained in the crypts or infective organisms entangled therein. Causes liable to be overlooked are intestinal infections and toxæmias, enteric fever, rheumatism, and pyelitis in girl babies. Acute coryza is obvious, except in the very early stages, but true influenza is often unrecognised, and still more often diagnosed on insufficient grounds. In making a diagnosis it is necessary to examine the patient for the onset of a specific fever, cerebrospinal meningitis, acute anterior poliomyelitis, pneumococcal infections, loculated empyema, osteomyelitis, otitis media, gastritis, and all possible causes of sepsis. Constipation alone may be the cause, and fæcal impaction may exist though the bowels act daily. Neurotic children are particularly liable to fever from excitement, undue exertion, and overfeeding. Chill and insufficient clothing are sometimes to blame, and at times one can ascribe the condition to heat stroke, malaria, tuberculosis, osteo-arthritis, or even hysteria.

Hyperpyrexia may occur in sepsis neonatorum, heat stroke, malaria, infective endocarditis, specific fevers, towards the end of meningitis, and, rarely, in rheumatism and chorea.

Occasionally fever persists for months, without any apparent cause and ending in recovery. Thus, a girl of 12 years had an irregularly hectic temperature, sometimes up to 104° F., and only coming down to normal in the course of five weeks; twice she had mild rigors. Puberty was



beginning, and possibly the fever was in some way connected with this. In another instance the fever persisted for about 2 years. I saw the girl at the age of 6 years for a hectic fever of 2 months duration, with loss of flesh and occasional epistaxis, and a history of cough and malaise for 4 months. Even at the end of 2 years, though plump and looking healthy, she was not entirely free from fever. A third girl, in her fourth year, of neurotic, artistic, and rheumatic parentage, was subject to attacks of fever of about 2 hours duration, her temperature rising to 103° or 104° F., and followed by excitement and a kind of epileptic fit. She was a mischievous and destructive child, but no cause for the fever could be found, except the unstable state of the nervous system. In a fourth case, also that of a girl, fever persisted for 10 months, as a sequel of mild typhoid fever, and ended in death from marasmus. In such a case there is most probably a chronic blood infection.

In practice cases of obscure fever are a source of great anxiety. High fever with little general malaise is less serious than the temperature suggests. More importance should be attached to the general state of the child than the actual height of the fever. Prolonged irregular fever necessitates repeated thorough examination to exclude all possible causes. Either a definite or the probable cause is found, or the patient gets well. The prognosis must be guarded, for it is clearly impossible to say in early stages that nothing beyond the fever will develop. It is wiser to state to the relatives that there is no discoverable cause, that they must wait with patience, rather than to jump at a doubtful diagnosis or make alarming suggestions of malignant endocarditis, tuberculosis, or other deadly complaint.

*General Treatment.*—The reduction of fever does not necessarily shorten disease. It is unusual and unnecessary to reduce the temperature, unless its height is such as to have an unfavourable result on the system as a whole, and the course of the disease in particular. Unfavourable indications are drowsiness, delirium, convulsions, a rapid and weak pulse, restlessness, and sleeplessness. The treatment depends on the height of the temperature, the duration of the fever, the nature of the disease, and its probable course. Severe remedies are not needed for high fever in a pneumonic attack near its crisis, but are imperative in hyperpyrexia due to rheumatism. Hydrotherapeutic measures, such as cradling, sponging, cold fomentations, cold packs, and a cold bath, lessen the intensity of the chemical activity by which the febrile temperature is produced, probably by increasing leucocytic activity. If there are cerebral symptoms apply ice or Leiter's coils to the head, or let it rest on a cold water rubber bag as a pillow. Rectal irrigation with ice-cold water is useful for infants and young children, if there is not much prostration.

Elimination is encouraged by saline purgatives, diaphoretics, and diuretics. The use of synthetic drugs, coal tar products (p. 82), is based



on a false idea that the malaise is due to the fever. For infants they are unnecessary and often dangerous. Phenacetin is the most harmless. Tincture of aconite, m.  $\frac{1}{2}$ -1 hourly for 8 doses is sometimes used, but is undoubtedly depressant. Salicylate of soda and quinine or euquinine are the best remedies. Quinine, in doses of 1 gr. per year of life, is very useful in late stages of broncho-pneumonia with a hectic type of fever and a failing heart. Dover's powder is given for irritability and restlessness. Alcohol may be used as a stimulant, and strychnine if there is depression. The diet should be light, liquid, and cool.

**Treatment of Collapse.**—Collapse is usually associated with a subnormal temperature, but the latter may occur without collapse. A subnormal temperature is due to exposure to cold, prematurity, malnutrition and inanition, fluxes and hæmorrhages; sometimes to heart disease, and frequently is a sequel of acute disease, such as specific fevers and pneumonia. In some instances, e.g., infantile cholera, the collapse is very analogous to shock, and may possibly be due to the action of toxins on the splanchnic area; or it may be due to cardiac failure, or the severe pain of colic, and the passage of calculi. In its mildest form it is treated by hot bottles, fomentations, packs or bath, and stimulants by the mouth. For great prostration, with cold extremities and pallor of the skin, give a mustard bath, or apply a mustard leaf or hot fomentations to the præcordium. A hot or mustard bath, with hot whisky and water internally and strychnine (s.o.s.) subcutaneously, is the best treatment for subnormal temperature due to cold and inanition. Subcutaneous injection of adrenalin m. 1-5 (1 in 1000) is said to raise blood pressure, but is probably useless unless given intravenously. It acts as a direct stimulant of the heart and peripheral vessels, causing contraction, and an excessive dose may cause rigor of the heart.

**Vaccine Treatment.**—The vaccine treatment of tuberculosis is similar in character to that of all microbial infections. According to recent views there is present in the serum a substance, called *opsonin*, which combines with the microbes and prepares them for ingestion by the leucocytes. Hence, phagocytosis varies directly as the amount of opsonin. This substance is destroyed by heating at 60° C. for 10 minutes.

Leucocytes washed free from serum have no phagocytic action on bacteria. Their phagocytic power remains constant. This is shown by taking washed leucocytes from a normal and a phthisical person, mixing them with serum from another normal person and an emulsion of tubercle bacilli, and incubating them. An equal phagocytosis results in each case. To obtain the *opsonic index*, mix an equal quantity of washed leucocytes, microbial emulsion, and the serum from a normal person and the patient. Incubate at 37° C. for 15 minutes, and make blood films. Count the number of organisms taken up. The opsonic index is the relative proportion of the numbers in the two cases. Thus, if 100 microbes are taken



up by 100 cells in normal serum, and only 70 in the patient's serum, the patient's opsonic index is 0.7. The opsonic index is, therefore, the measure of the resistance of the blood serum to microbic invasion. Raise it and the resistance to infection is raised.

The opsonic index to tubercle bacilli varies between 0.8 and 1.2 in normal individuals. It is lower in the infant than the mother. In localised tuberculous infections it is below normal, and in unlocalised ones it fluctuates from high to low. In slight early cases it is above normal; in chronic cases it is below normal; and in acute cases it fluctuates greatly from day to day. A high index may mean auto-inoculation. This can be induced by exercise, by manipulation of the affected part, and by suction cups, because of the increased blood supply. It is important to estimate the index before and after exercise to ascertain whether auto-inoculation is taking place, and to rest during treatment.

*Mode of Treatment.*—Isolate and cultivate the infecting organism. Estimate the patient's opsonic index for that organism. Raise the index by inoculating with vaccine, i.e., with dead organisms in salt solution. Re-inoculate during the maximum height or the fall of the positive phase, but not during the rise or in the negative phase. Each inoculation is followed by a *negative phase*, i.e., a drop in the opsonic index, which lasts for a few days, and is quite short in the healthy. In tuberculosis a negative phase may last from a few hours up to 14 days; and it may be preceded by a short rise, the *pre-negative phase*. By the *positive phase* is meant the gradual rise of the index to a maximum, immediately after the end of the negative phase. It remains there for a time and then slowly falls. There is little or no rise of temperature unless big doses are used. Injection during the negative phase causes a further drop in the opsonic index and diminished resistance. Negative phases can be accumulated one on another; not so the positive phases, but one positive phase can be followed by another with only a short and slight intervening negative phase.

In treating tuberculosis in children a dose of  $\frac{1}{50000}$  -  $\frac{1}{10000}$  mg. is injected, and repeated in increased strength every 10-14 days; the patient must be kept quiet for a day or two afterwards. The injection is of very little value if the index is already high. No injection should be given during intercurrent acute illness. The effects can be estimated by taking the opsonic index in 24 hours, 72 hours, 7 days, and 14 days, to determine the phases. Clinical experience shows that the injections can be given about every 14 days with safety, without troubling to estimate the opsonic index.

This treatment may be considered as still on its trial. It is spoken of favourably in cases of tuberculous adenitis, dactylitis and joint affections. The best results are obtained in staphylococcal infections, such as acne, multiple boils, and abscesses. At least 6 doses are required, often more, at intervals of 17-21 days.



**Anæsthesia.**—Anæsthetics must be given with as much precaution as in adults. Bear in mind the small size and compressibility of the thorax in infants, and the effect of fear in older children. Examine the mouth for loose teeth, sweets, and other foreign bodies. As a rule operations should be done at the time of the evening or midday meal, for children are not in such a good condition as adults after a night's fast. In *light anæsthesia* dangers arise from shock and laryngeal spasm. In *deep anæsthesia* suffocation may be due to the tongue falling back into the pharynx. The selection of the anæsthetic depends on the condition of the child, and nature of the operation, and its probable duration. Nitrous oxide gas, ethyl chloride, A.C.E. mixture, ether, and chloroform, are the usual anæsthetics, and are mentioned in the order of safety. Ethyl chloride and chloroform can be given during sleep. Chloroform is generally taken extremely well by babies. Children should be anæsthetised on the operating table.

*Nitrous oxide* gas is unsuitable for young children, especially if nervous, as the apparatus is alarming. They are frightened, and take it badly. It is suitable for short and trivial operations, such as tooth extraction, and as a preliminary to ether administration, in older children. It may cause inconvenient rigidity, jactitation, and involuntary evacuations. Being often followed by screaming and struggling it is unsuitable if a wound has to be dressed.

*Ethyl chloride* has largely replaced nitrous oxide, but is more dangerous, and is more often followed by vomiting, headache, and restlessness. It is pleasant, simple, easy to give, and does not irritate the air passages. The mode of administration is not alarming. Somnoform is a mixture of ethyl chloride 60 parts, methyl chloride 35 parts, and ethyl bromide 5 parts. It has no special advantages over the pure ethyl chloride, and is more expensive. Ethyl chloride can be given as a preliminary to ether or chloroform. It prevents the struggling and excitement induced by ether. If followed by chloroform, the latter must be given slowly, as its effect cannot be appreciated while the patient is under the influence of ethyl chloride.

The patient should be prepared in the usual manner. For babies and young children  $\frac{1}{2}$ -1 c.c. is sprinkled on lint and held over the mouth and nostrils. It must be given quickly. For older children a Clover's inhaler can be used, so that recourse can be had at once to ether if the operation is prolonged or the patient weak. A gag should be inserted for mouth operations, and the chin held well forward to allow air to enter freely. Consciousness is lost in 10-15 secs. The breathing becomes slow and deep, the pupils usually dilate, and the muscles are relaxed, except the masseters. In deep anæsthesia the muscles become, if possible, more relaxed, the face flushed and perhaps covered with sweat, the corneal reflex sluggish or absent, and an external squint often develops. There is no cyanosis or venous congestion. Unconsciousness lasts from 1-2 minutes. Recovery is



rapid; preceded by a dreamy state and slight mental confusion. An overdose is followed by headache, excitement, nausea, and vomiting.

This anæsthetic is not absolutely safe, for the rapidity with which anæsthesia is induced increases the liability to overdose and death from syncope or asphyxia. McCardie, in 1906, collected 21 deaths, of which only 3 were children, showing its comparative safety for children to whom it is so often given. It is especially suitable for short operations, such as the removal of teeth, tonsils, adenoids, circumcision, and the opening of abscesses. It must not be used for long operations, except as a preliminary. For the removal of tonsils and adenoids it can be given in the sitting position, but if either the operator or the anæsthetist be inexperienced the child may come round too soon, or too large a dose may cause danger from abolition of the cough-reflex or muscular rigidity.

*Ether* is an admirable anæsthetic for long operations provided plenty of air is given. Babies take it well. It has been credited unduly with safety, and insufficient importance attached to the subsequent bronchitis and pneumonia to which it may give rise. Fatal cases are generally due to one of these sequels. It is contra-indicated in the presence of respiratory troubles, and for operations on the abdomen or brain.

*Chloroform* can be given safely by the open method, if plenty of air is allowed, although the percentage of vapour to air varies within wide limits, from 1-10 per cent. With care the former percentage is the usual amount. For safety it should be continuously given in 1-2 per cent. strength, but the various inhalers used for this purpose are likely to alarm the child. Begin gradually with a few drops, and increase steadily by adding more at definite intervals. Methylated chloroform is not inferior to the ethylic variety, and is much cheaper. If it is not pushed beyond the stage of complete anæsthesia the percentage of drug within the blood is one within the limits of safety.

In the first stage there may be some preliminary agitation. Then sensation and voluntary movements are suppressed, while reflex and automatic ones are preserved. The corneal reflex is present. The pupils are usually a little contracted, but may remain dilated to the end of this stage; they react to light. This is called Light Anæsthesia.

In the second stage, that of Deep Anæsthesia or Surgical Anæsthesia, the reflex movements are suppressed and the corneal reflex abolished. Finally the automatic respiratory movements cease, and are quickly followed, within 60 secs., by stoppage of the heart, and death.

The corneal or lid reflex is elicited by touching the centre of the cornea or the conjunctiva, and feeling and seeing the responsive closure of the upper lid. The lid reflex may be absent, but the cornea give a slight response. As long as the latter is present and the breathing is free the patient is in a satisfactory state. It is not necessary to abolish the corneal reflex, and some anæsthetists regard its disappearance as indicative of the



stage of dangerous anæsthesia. It is unreliable in children, and on the whole the pupils are a better guide, for damage may be done to the cornea by frequently touching it with the finger, especially if wet with chloroform. The pupils should be contracted and react sluggishly to light. Dilatation may indicate reflex irritation at the wound, the onset of vomiting or danger. It may be present, extreme and fixed in some patients during deep anæsthesia, without danger if the breathing is satisfactory. If the pupils begin to dilate and the lid reflex returns, it indicates too little anæsthetic. A dilating pupil with loss of corneal reflex is generally a sign of danger. On the other hand the contracted pupil is not an absolute sign of security against respiratory failure. It is important to watch the respiration rather than the pulse. Breathing becomes shallow, irregular, and imperceptible from an overdose of the drug, while the pulse may be strong and regular, or may fail about the same time. A preliminary injection of atropin has been said to reduce the risk, but Webster's recent experiments on dogs contra-indicate this.

Chloroform and Ether, and A.C.E. mixture are pleasant, and safer than chloroform alone. They are given gradually and unconcentrated. An overdose is indicated by dilatation of the pupils and respiratory failure.

Anæsthetic deaths may occur before true anæsthesia is established, and the operation begun. Similar deaths occurred from fright in pre-anæsthetic days. They are unavoidable, but care should be taken not to alarm the child and to acquire its confidence. Apart from status lymphaticus (q.v.), death during operation is never quite sudden, and is due to gradual failure of respiration or circulation, from the action of the poison on the medulla. After operation death may take place within 10 minutes as a result of continued toxæmia, or from vomiting and aspiration of food into the larynx. Later on occur the cases of delayed chloroform poison described subsequently.

*Treatment of impending calamity.*—At once try and restore respiration. Rub the lips briskly with a dry towel, insert a gag, and pull forward the tongue. Rhythmical tongue traction is sometimes used, and is a somewhat barbarous method, as tongue forceps are needed. Lower the head and raise the feet; babies can be held head downwards. Try artificial respiration combined with inhalation of oxygen. Give a hypodermic injection of strychnine or ether. Apply heat in the form of hot blankets and bottles to the limbs, and hot towels to the præcordium. If there is much shock inject strychnine subcutaneously and saline infusion into the median basilic vein, and bandage the limbs from below upwards (auto-transfusion). For cyanosis with good heart sounds, give inhalations of oxygen or amyl nitrite. Acupuncture and massage of the heart are the last and most desperate remedies. Faradism can be tried.



## CHAPTER V.

### PREMATURE INFANTS.

An infant born only a week or two before term requires no special care or diet, provided it is well developed and weighs over 6 lbs. It is the infants born at an earlier period of gestation that are difficult to rear, and the difficulty increases in proportion to the diminution in maturity. Few born before the twenty-eighth week of intra-uterine life survive. Villemin reared one, who was born at  $5\frac{1}{2}$  months, or on the most liberal computation at 6 months, and weighed less than 2 lbs. In estimating the chance of life of a premature infant the degree of development is of more importance than the actual age, even if this could be ascertained. A premature infant is not always a weakling. No matter how small and delicate, an attempt must be made to preserve its life.

The premature infant is under weight, breathes and eats imperfectly, has incompletely formed organs, and ill-developed functions. The cry is feeble. The muscles are weak and the child may be absolutely inert, quite motionless and in a kind of torpor, and perhaps unable to suck on account of the muscular debility. The skin is soft and delicate, bright red in colour, loose and movable, with little subcutaneous fat and plentiful lanugo. After the seventh month the skin is not so brightly red. Hair may be present on the scalp. The nails may be absent, or thin, small, and not projecting beyond the finger tips. The centres of ossification of the lower end of the femur and upper end of the tibia are usually absent. The head is large, face small and wizened, belly protuberant, and the lower limbs less developed than the upper limbs. The brain is soft and ill-differentiated; the lungs anæmic, and presenting patches of atelectasis; the foramen ovale and ductus arteriosus patent; and the ductless glands large. Digestion, absorption, and assimilation are defective.

These babies are liable to sepsis, cerebral hæmorrhage and affections of the nervous system, gastro-enteritis, asphyxia, atelectasis, and pneumonia; hæmorrhages, because of deficient blood coagulability; abrasions of the skin; umbilical hernia, urinary infarcts, and sclerema.

Warmth, diet, and protection are the main essentials in treatment. Daily weighing is the only available indication of progress, and until there is a regular progressive increase in weight we cannot be certain that the child will live. The loss of weight, from 1-14 oz., during the first 3 days



depends upon the amount of urine and meconium passed, and the amount of water and food given. It may bear a much higher percentage ratio to the total weight than in a full-time child, and is more serious, for it continues longer and is more slowly regained. The mortality is greatest during the period of loss or stationary weight. The child who attains its birth weight in 2 or 3 weeks does very well.

The weight is no measure of prematurity, for it varies in infants at the same period of gestation just as it does in infants at term. The daily increase should bear some proportion to the original weight, and be greater in the heavier infants. At 20 weeks the average weight of the foetus is 39 oz. (Hecker and Lusk), and the daily gain in order to attain the same rate of development as in utero should be 0.75-1 per cent. of its weight. The mortality rapidly diminishes with the increase in the period of gestation.

Potel's Statistics.	Percentage Mortality.	Average daily gain in weight.
At 6½ months 11 survived out of 56	80.4	9.4 gms.
At 7 „ 55 „ „ „ 131	58.1	• 11.5 „
At 7½ „ 36 „ „ „ 53	31.0	13.8 „
At 8 „ 71 „ „ „ 110	35.5	22.8 „

Nearly 50 per cent. of the 350 survived. Weighing must be conducted with the greatest gentleness, for the child should be handled as little as possible.

The gastric capacity is estimated at 1 per cent. of the body weight, and the composition of the food is the same as that for ordinary infants, but a weaker quality. Weakly babies must be fed hourly. Maternal nursing is often injurious as lactation is not fully established. A wet nurse, who has been confined at least a month previously, is preferable to the mother if the child is strong enough to nurse. She should nurse her own child in addition, and give the last part of the feed to the foster child. If suckling is out of the question from ½-1 teaspoonful of a 5 per cent. solution of milk sugar may be given hourly for 24 hours, and then a similar quantity of the breast-milk of a wet nurse added to each feed and the amount gradually increased. Or recourse must be had to whey, weak peptonised milk, weak condensed milk, or a laboratory mixture containing casein 0.25, albumin 0.25, fat 1.0, and sugar 3.0 per cent., one teaspoonful being given hourly. Or a mixture of cream, lactose and water can be used. The quality and quantity of the food are increased according to the way in which it agrees. The chief point in feeding these babies is to give a very weak fluid, containing little fat, in small feeds hourly. This reduces the risk of dilatation of the stomach and gastro-enteric troubles. Insufficient diet is indicated



by feeble continuous cries. When taking enough, the child sleeps quietly between each feed, but the lethargy due to debility and inanition must not be mistaken for contentment. With satisfactory progress the interval between the feeds can be increased.

If the child is too feeble to take an ordinary feeding bottle, it should be fed by means of a glass cylinder, graduated in cubic centimetres or half drachms, with a sloping end to which an ordinary teat is fixed, an india-rubber ball being attached to the larger end. The warm food is poured into the cylinder, the rubber ball fixed on, and the food forced slowly into the child's mouth. This method is especially suitable for a baby kept in an incubator, for the child need not be handled. If the baby is not able to swallow, it must be fed by means of a tube passed into its stomach.

The temperature falls rapidly to 90° F., or lower, unless special care is taken, and is subsequently very unstable. It is of the utmost importance to maintain the temperature near the normal level, by preventing the loss of heat as well as by diet. The child should be wrapped in layers of cotton wool, covered with blankets, and kept in a cradle, containing 2 or 3 hot water bottles or 1 U-shaped one, or electric heaters. The room temperature must be maintained at 90° F., and all changes of clothing made before a good fire. Unfortunately this temperature is very trying to the nurse, that of the cradle cannot be absolutely gauged or regulated, and the frequent changing of the napkins exposes the baby to considerable risk.

Various incubators, hatching cradles, or brooders have been devised, and one such apparatus should be attached to every maternity home or hospital. A useful one can be bought or hired from Hearson, Regent Street, London. An incubator should be simple, reliable, easily cleaned, and easily regulated as to the supply of heat, moisture, air, and light. If the child is very premature the temperature should be kept up to 90-95° F., and gradually reduced as the child gets older and heavier, e.g., to 68° F. at 5 lbs. weight. A temperature of 80° F. is often high enough. In all cases it must be lowered if the child sweats, is restless, cyanotic, breathes rapidly, or its temperature rises. The air supply should be pure and contain 60 per cent. of moisture. The incubator must be kept thoroughly clean, well ventilated, and not exposed to the direct rays of the sun.

Washing should not be allowed if the child is very weak, and even the bath at birth must be omitted. If the child is rubbed over with warm oil and kept in an incubator, washing may be postponed for a couple of months. If a bath is given, the water should be previously boiled and a superfatted soap used. A bath at blood heat and raised to 105° F. is a useful means of raising the temperature, if much subnormal.

No clothing is necessary beyond cotton wool, which should be changed morning and evening. A separate piece can take the place of a napkin, and be changed when necessary. Drying powders may be used in moderation, and warm oil relied upon for cleaning purposes.



*General Directions.*—Maintain the temperature of the child, and take its rectal temperature regularly. Carefully regulate that of the incubator. Give a suitable diet and brandy if necessary. Avoid handling and all measures likely to cause exhaustion. Guard against exposure to light and noise, and infection of the alimentary or respiratory tract. Nasal catarrh is a serious complication. Note the character of the cry, the presence or absence of colic, and the character of the stools, for the crying may be due to hunger or indigestion. A marked fall in temperature, coldness of the hands and feet, and increased frequency of respirations may indicate that the apparatus of the incubator is wrong or that the food supply is deficient. A continuously low temperature is often due to low vitality, and irregularity to lack of control of the heat regulating mechanism. A sharp rise may be due to too high a temperature of the incubator, septic infection, and gastro-intestinal or pulmonary complications. Oxygen gas is useful if there is cyanosis. Brandy is constantly necessary, and occasionally recourse must be had to saline injections.

*Prognosis.*—Few born before the twenty-ninth week survive. Those under  $2\frac{1}{4}$  lbs. in weight generally die, and those over  $3\frac{1}{2}$  lbs. usually survive. Even when a child is gaining weight, death may occur absolutely suddenly. Rapid loss of weight, overlapping sutures, and a rectal temperature below  $90^{\circ}$  F. are very bad signs. A continued slow loss of weight, subnormal temperature, and apathy are unsatisfactory symptoms. Breathing is usually irregular, imperfect, and may assume the Cheyne-Stokes type. Attacks of cyanosis are the commonest cause of death, and result from atelectasis or failure of the respiratory centre. Pulmonary collapse is frequent and very fatal during the first 2 or 3 days, but rare after the tenth day. The longer the child lives, the less severe are the attacks of cyanosis, and the less likely are they to be fatal. They may be due to food getting into the trachea. Sudden death is often due to cardiac failure.

General œdema is common on account of the feeble heart, anæmia, and imperfect action of the kidneys. Many of these babies are very anæmic, because of the delayed development of hæmoglobin formation. Nasal catarrh, bronchitis, and gastro-enteritis are serious complications.

The most favourable conditions are two skilled nurses, an incubator, a physician who understands such cases, and parents who do not interfere and press for changes of treatment. Eventually these babies develop into normal children. By the use of the incubator and forced feeding the mortality has been reduced 50 per cent. for premature infants born at  $7\frac{1}{2}$  months, and about 20 per cent. for those more premature.



## CHAPTER VI.

### MORTALITY.

*Intra-uterine death—Intra-uterine putrefaction—Rigor mortis in the stillborn  
—General mortality—Death from overlaying—Sudden death.*

Though the incidence of life is associated with the liability to sudden or unexpected death, and the fragility of the hold on life in the first months of existence is great, the death of a child after this period should be regarded as unnatural.

**Intra-uterine death** may take place quite suddenly, even within half-an-hour of birth. The foetal movements cease. It may be due to malformation of the heart or main vessels, premature closure of the ductus arteriosus, or serious congenital defect. The left lung was absent in a case of Schrader's. Sometimes it is due to disease or separation of the placenta, or to injury. Occasionally no cause can be found. A child born in a state of apparent death is not necessarily dead, so the usual means of resuscitation must be tried, viz., rhythmical traction of the tongue, insufflation, etc.

**Intra-uterine putrefaction** is nearly always caused by foetal decomposition. In exceptional instances a live child has been born, the putrefaction being due to decomposition of the placenta or of blood clot.

**Rigor Mortis** can occur in a stillborn child before or very shortly after birth, so it must not be regarded as reliable evidence of live birth. Chowne published the first ante-natal case in 1841, and a good many have been recorded since. One of twins has been affected, while the other lived (Ulrich). This ante-natal rigidity may pass off before labour; may prevent labour until it has passed off; may hinder labour, and give the sensation of a half-frozen child to the examining finger; may continue after delivery. It may set in during or after confinement, the foetus having died in the late stages. It does not always accompany still-birth. The rigidity varies in degree, is most marked in the upper limbs, and causes the body to assume the intra-uterine position. The head is drawn forward, back somewhat rounded, arms flexed at the elbows and bent on the chest, and the thighs and legs flexed. More rarely the body is fully extended and rigid "like a soldier at attention," in the cadaveric position, and in this form it has even caused fatal injury from rupture of the uterus, due to the feet passing through its wall. Usually the cadaveric position in rigor mortis



implies that the child has had a separate existence, and the intra-uterine position implies still-birth. This must not be accepted as universally true. Ante-natal rigor mortis may be associated with hæmorrhage. It does not return after passing off.

**General Mortality.**—The death rate of infants is lowest among the British Peerage, and the fisherpeople of the Farøe Islands. It is lowest in the breast-fed, except in places like Bavaria, where the infants are taken by their working mothers to the fields and exposed to cold and wet. It is highest in the bottle-fed infants in manufacturing centres, where the mothers work in factories. It is greater, for instance, in the northern than the southern parts of Staffordshire, for the latter is a mining district, and the women remain at home, while in the former the women work in factories as potters. Infantile mortality depends on defective viability of the child through ante-natal conditions affecting the mother, such as insufficient food, anæmia, alcohol, overwork, lead and other poisons, insanity, and constitutional disease. It may be due to injury at birth, asphyxia, or intracranial hæmorrhage; or it results from post-natal conditions, many of which are indirectly due to ante-natal causes, such as debility, inanition, atrophy, marasmus, and convulsions; prematurity; asphyxia, atelectasis, syphilitic pneumonia, and congenital defects.

The number of premature infants has shown a considerable increase in recent years, the cause of which is uncertain. Perhaps it is due to the survival of the delicate up to parental age under the improved environment of modern life; to the use of drugs and other means to procure abortion; or to artificial measures adopted to prevent conception, leading to minor effects on the endometrium. Both prematurity and still-birth are more frequent in boys than girls, and in the illegitimate than the legitimate, except where illegitimacy is not considered a disgrace.

Infantile mortality is very high in the first month, sinking to less than half in the second month, and progressively decreasing with each successive month during the first year. It decreases with every day of advancing life, for the younger the infant the greater are the effects of ante-natal debility, of injury at birth, and of congenital defects, and the more active are the processes of disease.

Death during the first year is ascribed to congenital weakness in 15 per cent., infections in 5 per cent., and respiratory diseases in 10-20 per cent. One-third may be put down to disorders of the digestive organs, and one-third to various other causes. Alimentary affections are the chief cause among the poor, increasing in frequency with the poverty of the parents, and comparatively rarely fatal among the well-to-do. In the second year of life about three-fifths of the deaths are due to respiratory and one-fifth to alimentary troubles. A marked rise or fall in the atmospheric temperature increases the mortality. Especially is this the case with a high summer temperature and diarrhoeal affections in large towns. Cold lowers



the vitality, and leads to fatal affections of the respiratory organs. The neglect of suckling is one of the chief causes of death in infancy.

Poverty, leading to the dependence of the child on the maternal milk supply, reduces infantile mortality. No woman should be allowed to return to any work, which interferes with regular nursing of her infant, until 3 months after confinement.

**Death from Overlaying.**—Nearly 1,500 infants are killed yearly in England and Wales through being overlaid by one of the parents. Death results from suffocation by the pressure of the arm or body. Occasionally it is due to the pressure of another child, the domestic cat or dog, or of clothes. Or the child may turn on its face, and bury the mouth and nostrils in the pillow. In most instances this avoidable cause of death must be ascribed to carelessness, reckless indifference, or culpable neglect, almost amounting to manslaughter or murder. Drunkenness in the parent, and the lack of a cot or cradle for the child are the two most important factors. A padded soap box or a hammock would be quite sufficient. No child should be permitted to sleep in the same bed as a parent until 2 years of age.

Drunken parents who cause the death of their offspring in this way should be severely punished. More than three-fourths of these babies are under 3 months of age. After 6 months of age comparatively few succumb, and over 1 year the child is generally strong enough to protest.

Examination of the body may show marks of pressure, e.g., a flattened nose, or merely signs of suffocation. Asphyxia occurs from other causes, so cannot be accepted alone as proof of death from overlaying. Usually there are found bluish lips and livid face, reddened eyeballs, punctiform conjunctival hæmorrhages, sometimes a protruded and discoloured tongue, and froth, often bloodstained, in the mouth or nostrils. The lividity of the complexion may soon pass off. The limbs are flexed, the hands clenched, and the nails blue. The blood is dark and liquid, the right ventricle distended with soft clot and the left contracted and empty. The mucous membrane of the trachea is reddened. The lungs are congested, sodden, and œdematous, containing much frothy fluid, but yielding no microscopical evidence of inflammation, in contra-distinction to that of acute pneumonic congestion. Minute petechiæ are found on the serous membranes. The brain and meninges are congested.

**Sudden Death** is of frequent occurrence, and requires special study in connection with those cases which do not lead to the child being placed under medical care. Strictly it must be ascribed either to failure of the circulation, or of respiration, but it will be more convenient to discuss it under the headings of syncope, asphyxia, convulsions, intestinal disorders, and toxæmia. Some of these are organic, others apparently functional in origin. An unexpected death, in the course of a disease which seems to be running a harmless course, is also a sudden death, lamentably so from the parent's point of view unless the possibility thereof has been suggested.



Syncope or heart failure is the common termination of many diseases, especially prolonged wasting affections. Many a marasmic infant dies suddenly from this cause, with or without slight agonal convulsive movements. Sometimes the cause is a myocardial weakness dependent on fever, a cloudy degeneration, a fatty change, atrophy of cardiac muscle, an unsuspected myocarditis, or fibroid disease. Death is myocardial in origin when it ensues on slight exertion in pneumonia, diphtheria, influenza, and other specific fevers. The heart may exhibit severe myocardial changes, though to the unaided eye appearing normal. Less frequent causes are congenital and acquired heart disease, adherent pericardium, pericardial effusion, acute dilatation of the heart following extreme exertion or whooping cough, the displacement of the heart by rapid pleural effusions, and possibly the cumulative effects of digitalis. Spontaneous rupture has followed septic embolism and abscess of the myocardium, even in the first year of life.

Syncope may occur from a fit of coughing or from violent pain, reflex action or direct inhibition. Death from fright or excitement is not unknown, and is almost certainly due to vagus inhibition of the heart. Inhibition may be caused by a slight blow or pressure on the larynx, a blow in the epigastrium during digestion, a blow in the hypogastrium, sudden injury in the regions supplied by the trigeminal nerve, a sudden cold plunge, minor operations, e.g., circumcision, puncture with a trocar or exploring syringe, the hypodermic injection of serum, or the sting of a bee. Death by cardiac inhibition is sudden, calm, and unaccompanied by convulsions. Cervical caries, with separation of the odontoid process, might cause it by pressure on the cord. The sudden lowering of blood pressure by the rapid escape of cerebro-spinal fluid may be quickly fatal.

In many of these cases of sudden and fatal syncope there is another important factor present, namely, the status lymphaticus (q.v.). It is incredible that several of the slight causes mentioned could produce the fatal result, were it not for the presence of some peculiarity of constitution which renders the child extremely susceptible to the effects of shock.

Asphyxial causes include glottic spasm from laryngeal irritation by foreign bodies or inflammation, obstruction in some portion of the respiratory tract by too large a bolus, or by food regurgitated and inspired when the child is on its back, from a smack on the back while eating, during artificial feeding, or in post-anæsthetic vomiting; or by various foreign bodies, a round worm, the contents of a tuberculous cavity, or a caseating gland ulcerating into the trachea. Acute periglandular œdema and swelling may cause compression of the trachea and death in a few hours from suffocation.

Acute pulmonary congestion or inflammation, atelectasis, the rapid effusion of a pleural effusion and laryngospasm may all end fatally. Cardiac



thrombosis is an occasional cause. A thrombus may be carried into the pulmonary artery from the lateral sinus in ear disease.

Death from asphyxia is rarely as sudden as that from syncope, and some of the pulmonary causes act rather through the nervous than the respiratory system. Thus the so-called "inward convulsions" are often due to glottic spasm of purely nervous origin. Analogous spasms may be due to pertussis, to laryngeal papillomata or foreign bodies, or the pressure of enlarged glands on the vagus or its branches.

In asphyxial cases, especially those of acute capillary bronchitis, the "suffocating catarrh" of Laennec, liable to occur in delicate infants in the first 6 months of life, froth is found in the bronchi. Portions of the lung are hard, but float in water, and yield little rods of muco-pus on pressure; and subpleural ecchymoses are present. Similar congestion may occur in any fever, unsuspected typhoid and tuberculosis.

Convulsions are fatal from syncope or from asphyxia, due to spasm of the respiratory and laryngeal muscles. Hæmorrhage into the cranial cavity is a common cause at birth. Subsequently it may be due to blood diseases, toxæmic affections, or rupture of a blood vessel. Miliary aneurisms have been found in quite young children. Hæmorrhage is especially likely to cause sudden death when it takes place in large amount into the lateral ventricles.

Meningitis, cerebral tumours, the rupture of an abscess, and brain softening may all cause fatal convulsions. If there is fever with convulsions the onset of an exanthem should be suspected. If there is no fever, in nine out of ten cases the cause lies in some affection of the alimentary system.

Intestinal disorders, causing sudden or unexpected death, may do so through setting up convulsions, from profuse hæmorrhage, profound toxæmia, or the very rapid loss of fluid in cholera and cholera nostras. Death takes place rapidly but rarely quite suddenly.

Toxæmic states are best illustrated by the rapidly fatal issue in some cases of malignant fevers, cerebro-spinal meningitis, pneumonia, appendicitis, pyæmia, and others of uncertain origin. A rapidly fatal disease has been reported as occurring in institutions. Dr. C. J. Macalister described four such cases. A boy was sent to bed on account of an irritable cough, and was found dead half-an-hour later. Another had violent vomiting and diarrhœa, soon became profoundly weak, livid and unconscious, and died in 3 hours. A third complained of headache at night, was very weak in the morning, became unconscious, and died, with the appearance of asphyxia or strangulation. In a fourth child there was vomiting, a little diarrhœa, and a similar asphyxial appearance after death. Probably the exciting cause was a toxin acting on the respiratory centre, causing engorgement, œdema, and even pneumonia of the lungs. Suprarenal hæmorrhage has been found in cases fatal in a few hours from vomiting, diarrhœa, lividity, and collapse. It may occur in healthy robust children, and is



probably toxæmic. Death may follow the rapid cure of a skin eruption, e.g., the so-called "eczema death." Possibly in some of these cases a nephritis is set up, and in others the status lymphaticus is present.

Sudden death also occurs from anæsthetic and surgical causes. Shock may be due to or accentuated by cold during an operation; the rapid evacuation of a pleural effusion or psoas abscess; mere exploratory puncture; or the washing out of cavities. Death may follow the entrance of air into veins or embolism, for instance, after the injection of iron into a nævus.



## CHAPTER VII.

### CONGENITAL DEFORMITIES AND INJURIES AT BIRTH.

*Deformities — Ante-partum Injuries — Caput Succedaneum — Cephal-hæmatoma — Cranial Depressions — Sterno-mastoid Tumour — Diaphragmatic Hernia.*

Deformities have already been shown to be in many instances the result of inheritance, depending on the direct transmission of some peculiarity in the male or female germ cells. Others are ~~due~~ to injury, toxic or microbial infection, or poisons during intra-uterine life; or to actual intra-uterine disease, though this is more likely to cause abortion. Many defects, e.g., dimpling, grooving, and scarring of the skin, and amputation of limbs, are ascribed to amniotic adhesions or to strangulation by the cord. Malformations may affect every structure in the body. Many of these are referred to in the chapters on special diseases. Here a few of the more obvious ones will be described. Family deformities may be limited to one sex. Some are due to defects, others to excess of normal development.

*Acephaly* is a deformity incompatible with life. In the variety, *acephalus monobrachius*, there is only one upper extremity. In *para-cephalus* both upper extremities are absent. *Myacephalus* is a shapeless mass covered with skin. Deformities of the limbs may be present. There may be no rudimentary head; no trace of a cranial bone; a soft nodular mass of vascular connective tissue; or sometimes a circular line of hair and central depression at the upper end of the foetus. The cerebrum and cerebellum are absent. It is due to agenesis in early foetal life.

*Ectromelus* is ascribed to intra-uterine amputation of the arms and legs. It is most unlikely that the cord could exert sufficient pressure without its circulation being stopped. The only case, which has come under my care, was a boy, 3 months old, the fourth child of apparently healthy parents. The mother stated that she nearly aborted after a fright when 3 months pregnant. There were no deformities in other children. The patient was born at full time, and was well grown. The right arm was absent at the elbow, the left at the wrist; the right leg at the ankle, and the left at the knee. He died in a few days from infective diarrhœa. Well known cases are those of Matthew Buchinger and Arthur Kavanagh. Buchinger was born in 1674 and died in 1722. He could play several musical



instruments, write, draw, juggle, and shave; was married four times, and had 11 children. Kavanagh could write, draw, ride, drive, shoot, and fish, and was a member of Parliament. This deformity is not transmissible and not incompatible with prolonged life. All the extremities may be almost entirely absent. Bones can be felt in the stumps. Cases in which rudimentary extremities are attached to abortive or shortened limbs are clearly of a different type in that they cannot be due to intra-uterine amputation; they can be produced by abnormalities of development of the limb segments. There is often no history of shock or maternal impression. The extent of the amputations vary. It may be limited to one finger. Occasionally it is incomplete, the limb remaining attached by a fibrous cord, or simply marked by a line of constriction. The remains of a supernumerary little finger may be indicated by a little nodule near the base of the normal finger, as if amputated in utero.

*Split Hand and Foot* or *Lobster Claw* is a truly hereditary defect. The foot malformation is the most constant, so *Hereditary Split Foot* is the best name. It must be regarded in its origin as a sport or mutation, though it varies in degree in different individuals. Lewis and Embleton (1908) collected over 180 cases. In six generations of one family 44 were affected, generally in all extremities. The hands were never deformed alone, nor one foot alone. The feet showed three types of deformity: absence of the second, third, and fourth phalanges and metatarsals; deformity of the second toe chiefly; malformation of the third toe. Syndactyly of the unaffected toes was common. The feet have a cleft or bifid appearance. In another family of 87, 28 were affected (Robinson and Bowen, 1909).

Possibly this is an atavistic reversion. So, too, the peculiarity of an extremely short first metatarsal bone, inasmuch as it resembles that of an anthropoid ape in which the big toe is like a thumb. It may be bilateral. The whole foot indicates a reversion towards that of the higher apes. In the foetus the great toe is apposable, and in childhood it is much more movable than in later life.

In *Brachydactyly* the middle phalanges of the hands and feet are abortive, sometimes reduced to a cubical bone, generally ankylosed with the ungual phalanx. The first phalanx of the thumb is affected, and is, therefore, regarded as homologous with the middle phalanx. In Matthew's family (1908) it occurred in 12 members in five generations, was transmitted by affected females, and rarely affected males.

*Polydactyly* is not uncommon, and may affect all the extremities.

*Syndactyly*, or *Webbed Fingers and Toes* is still more frequent, and often associated with polydactyly or other deformity. Morrish (1907) reported the case of a man with an extra digit on each hand and foot, and webbing of both second and third toes. He was an only child and his parents were unaffected. Of his seven children two boys were normal; five girls had extra ulnar digits on each hand, attached by a pedicle or with bony union. Various



degrees of webbing of the toes were present, and three children had extra toes. Webbing of the fingers, generally between the third and fourth, is the most common malformation of the hand, and is due to an arrest of development. In early foetal life the fingers are bound together by webs of varying extent. The thumb becomes detached in the eleventh week, and the fingers separate later. Hence it is common for the thumbs to remain free, though all the fingers are united. In three recorded instances the thumbs were affected. Syndactylism of the toes is similar in character. The treatment is purely surgical.

*The Thumb and First Metacarpal may be absent.* This rare deformity is usually associated with defect or absence of the radius. Occasionally the radius is normal. The trapezium and scaphoid may be absent. An absent radius may be associated with club-hand. *Bifurcation* of the terminal phalanx of the thumb assumes two varieties. The bone may be bifid, and the nail single, though grooved. Or both may be bifid and united by a web. It may affect one or both thumbs, and both big toes. Other ungual phalanges may show similar conditions. Malcolm (1908) records a curious case in which the nails were absent on the thumb, first and second fingers, and radial half of the third. The feet were affected in the same way. It had occurred in four generations. Absence of the proximal phalanges, the fingers ending in rounded stumps, has been transmitted through five generations (Lewis Marshall, 1903).

Of the various deformities of the leg we find absence of the tibia, alone or associated with other defects, such as supernumerary digits and absent radii. In a case of Openshaw's the upper fifth of the tibia in the left leg was alone present, and the right external malleolus was absent. Both the fibulae and certain toes may be absent; or one alone, in conjunction with the outer half of the foot and two outer toes.

The patellae may be absent causing genu recurvatum, or congenitally dislocated, producing genu valgum. Possibly this is due to weakness of the vastus internus muscle.

**Ante-partum Injuries** are comparatively infrequent for the foetus is well protected. Nevertheless both slight and serious damage may occur, even though there is no external evidence of violence. In rare cases the child is born with a wound in process of healing, due to an accidental or intentional blow on the pregnant womb. Sometimes multiple fractures are found, but these are more likely to be the result of osteogenesis imperfecta.

*Injuries at Birth* are much more frequent, and due to the relative size of the head of the child, and the bony outlet of the pelvis; deformity of the pelvis; tumours; injury by the examining finger; and to the amount of violence, necessary or unnecessary, used in delivering the child, or to the local pressure of the forceps. The cranial bones may be fractured; the parietal being the most liable in unassisted labour, and the frontal if forceps are used. Fracture of the vertebral column; of one or more bones of the



limbs, notably the humerus, clavicle, and femur; separation of a humeral epiphysis; and single or multiple dislocations may all occur. Visceral hæmorrhages are sometimes found. Thus, we may find abrasions, ecchymoses, wounds, fractures, dislocations, internal hæmorrhages, rupture of internal organs, and even avulsion of a limb. Normally the cranial bones overlap, remaining so for a few days, and the skull is asymmetrical in consequence of the moulding of the head during its birth. This asymmetry is often distressing to the mother, if the child is a girl, for it persists for a long time, and, indeed, is more or less permanent in a very modified degree, insufficiently to be noticeable in later life. It is always safe to state that it is of no importance, and that it will gradually improve as the head increases in size. No head is truly symmetrical. Certain types of deformity of the skull will be considered later for they are not due to injury at birth, but to developmental errors. Much injury, even fractures, may occur in unaided labours. Injuries found after instrumental labours are by no means always due to the instruments used, and, even if they are, they may have been unavoidable.

The **Caput Succedaneum** is very commonly present. It is situated on the head, usually over the posterior part of the parietal or the membranous part of the occipital bone, but may be found on the nates, scrotum, or other presenting parts. It varies in size and shape; generally round, oval, or somewhat elongated. The superjacent skin is unaltered or congested, even purple, according to the duration and effect of the pressure. The swelling is due to a ring-like compression of the part first presenting, and serous or sero-sanguineous transudation into the cellular tissues. If many blood vessels give way the amount of hæmorrhage may produce a fluctuating tumour. It differs from a cephal-hæmatoma in its doughy or œdematous swelling, generally pitting; its greater diffuseness, not being limited by the sutures; and its occurrence at birth, though, if due to thrombosis, it may take 1-3 days to develop. Sometimes two are present, one being due to constriction by the neck of the womb, and the other at the pelvic outlet. No treatment is necessary, unless the skin is abraded or broken, or inflammation and suppuration ensue. Sloughing of the scrotum has been recorded. The swelling subsides in 1-3 days, and usually disappears entirely in a week.

A **Cephal-hæmatoma** is a special variety of tumour due to hæmorrhage between the periosteum and the cranium. Occasionally the term is used to include hæmorrhages between the scalp and periosteum (hæmatoma simplex), between the skull and dura mater (cephal-hæmatoma internum), and meningeal hæmorrhage. On the whole it is best to reserve the name for the type here described. Shortly after birth, generally on the second or third day, a slight swelling is found on the head, and it gradually increases in size for a few days to a week.

It is due to bleeding from the subperiosteal vessels or those passing



through bony foramina, in consequence of excessive moulding of the skull, injury by forceps, increased blood pressure, constriction of vessels of the scalp during labour, or asphyxia. The periosteum is raised by the hæmorrhage. Dark fluid blood and some fibrin are found between it and the bone, with bony formation later. The superjacent skin is movable over the surface of the tumour, and usually not discoloured. This tumour is most common in difficult or prolonged labour, and in vertex presentations. It may be found after spontaneous easy confinement, and in breech presentations. Eighty per cent. are said to occur in primiparæ, elderly ones being particularly liable. Of six successive cases all were in multiparæ; the fourth to the fourteenth labour. One was a twin. It is twice as frequent in boys as in girls, because of the larger size of the head. It occurs in 1 per cent. of all infants (Kleinwachter, 0·4-0·5 per cent.). A similar hæmatoma may result from injury after birth.

Though usually single, bilateral and even triple tumours are sometimes seen. The most common situation is over the right parietal bone, because of the frequency of left occipito-anterior presentations. In my experience it is just as common over the left parietal bone, but is rare over the occipital. In other situations it is more likely to be due to injury by forceps than to the other causes mentioned.

The swelling is soft, fluid, fluctuating, irreducible, painless, and free from heat and inflammation. It does not pulsate, and does not increase in size on cough or crying. In size and shape it is about that of half a hen's egg or a small apple, and it contains about an ounce of blood. It may be much larger, and hold 5 or 6 oz. Roughly it corresponds with the shape of the bone, for it is almost invariably limited by the attachments of the periosteum, and does not extend on to the sutures or fontanelles.

Though at first it may be tense, it is usually quite soft when brought for diagnosis. Gradually it hardens by coagulation of the blood, and presents a soft central area with a raised hard encircling ring. At the end of a week bony spicules and plates develop from the inner layer of the periosteum, spread into the encircling ring and over the whole surface, giving rise to a peculiarly crackling feel on examination. Sometimes the new bone is only formed at the edges, the fluid blood is absorbed, and the periosteum becomes adherent again. Unless the effusion is quite small, the ossification proceeds until a smooth, sometimes uneven, hard, bossy tumour is left, creating a certain degree of asymmetry or deformity of the skull. Ordinarily the tumour is well in 1-3 months, except for the bossing which lasts for as many years, eventually disappearing and leaving no trace. The absorption is due to the ordinary processes of growth in the periosteal bones.

Inflammation may result from severe traumatism. Infection is liable to lead to abscess formation, caries of the underlying bone, and secondary purulent meningitis.



This head tumour must be diagnosed from caput succedaneum, various forms of meningocele, dermoids, and fractures. A caput succedaneum is not limited by sutures, has no bony ring, and begins to disappear on the second or third day of life. A meningocele issues from a fontanelle or suture, pulsates, increases in size on cough and crying, is partly reducible, and causes cerebral symptoms when compressed. A cephal-hydrocele is associated with fracture and pulsates. Dermoids in this situation are very rare. The most common error is that of ascribing the tumour to fracture of the skull, because of the prominent hard ridge and soft centre. It is, however, a tumour, and not a depression, and the bony cranium can be felt on pressure in the centre of the swelling. Occasionally it is associated with a cranial depression or fracture. In one case hæmatoma of the left parietal bone was associated with a smaller subdural hæmorrhage, a similar subdural hæmorrhage on the opposite side, a frontal meningocele, and a myelo-meningocele in the lumbar region.

No treatment is needed, except protection from pressure and injury. Painting with equal parts of flexile and non-flexile collodion may help absorption by causing contraction. Evacuation by puncture and subsequent compression are unsatisfactory, and there is risk of sepsis. It may be advisable to evacuate the blood if the effusion is so great and rapid as to threaten the nutrition of the scalp; or if the skin becomes red and œdematous. No such case has come under my notice. Complications, such as fracture, intracranial hæmorrhage, and abscess, require appropriate treatment.

*Injuries of the face and head.*—Abrasions, bruises, and various wounds may be caused by the finger of the accoucheur, the pressure of forceps, other instruments, and falls during precipitate labour. Occasionally the pressure may be so severe as to result in local gangrene. Such a case came under my notice, although premature labour was induced in the eighth month because of the small size of the maternal pelvis. A patch of dry gangrene developed in front of the ear. The child showed remarkably little vitality, and died later from sepsis. Sloughing of the scalp, more or less severe injury to the eye, facial palsy, and dislocation or fracture of the lower jaw have all occurred.

**A Cranial Depression**, spoon-shaped or furrow-shaped, is due to pelvic deformity, the head being compressed against the sacral prominence or other bony ridge; and sometimes to instruments. It can be produced by persistent pressure with the finger in an attempt to turn, or by pressure against an elevated arm in a breech presentation.

It may occur in more than one child in a family. The common site is the frontal or parietal region, especially the left frontal, near the anterior fontanelle. Occasionally it is bilateral. About one-third of these children are stillborn. Some are asphyxiated at birth. Some die from twitching, convulsions, and paralysis.



The spoon-shaped depressions are the more serious. Frequently they give rise to no symptoms and disappear in a week or two, leaving no ill-effects. Some are permanent, though producing no symptoms or merely minor nerve storms. Others cause pressure symptoms, e.g., signs of irritation of the motor area. The frontal ones are more serious than the parietal. Out of 65 cases of spoon-shaped depression collected by Schroeder 32 died, 22 being stillborn, from the more or less direct results of the injury.

The depression may be associated with fracture, and perhaps subdural hæmorrhage. Fracture, usually parietal, may occur in either spontaneous or assisted delivery, even though there be no pelvic disability.

Many disappear spontaneously. Gentle manipulation, as by anterior and posterior compression of the skull, is of assistance. Possibly the application of a cupping glass is justifiable. If there are signs of compression and irritation, operation is essential. An incision is made through the adjacent bone, and the depressed portion is elevated by means of a periosteal elevator, inserted between the bone and dura mater. Or a disc-shaped piece of bone is cut out by the trephine, inverted and replaced. It becomes firmly re-united in a week or two. If it is removed, new bone will be reproduced in about 3 months. Trephining is more reliable than elevation, for, unless the elevation is done soon after delivery, the bone usually falls in again, and has to be removed.

A similar depression may be produced after birth, commonly in the parietal region, by falls and other injuries. Localised fractures also occur, and sometimes rupture of the dura mater, remarkably adherent in infants, with escape of cerebrospinal fluid and the formation of a *Cephal-hydrocele*. This is a pulsating tumour, which may be translucent. In fractures and falls, whether at or after birth, the injury to the brain is greater than in adults because of the yielding character of the bones and the ununited sutures. Yet the symptoms are slighter; cerebral compression is less readily produced.

**Sterno-Mastoid Tumour.**—(Hæmatoma or Induration of the Sterno-Mastoid). First described by Dieffenbach in 1830. Both sexes are equally liable. It is most common in first-borns (9 out of 14 cases). The tumour is found, usually accidentally, in one or other of the sterno-mastoid muscles about the second or third week of life, or even a few days after birth. Out of 13 successive cases 11 were on the right side. It is rarely bilateral. The common situation is at the junction of the middle with the lower third of the muscle, mainly on the inner side. At first the lump is soft, like fluid, but it gradually hardens and forms a nodular, ovoid swelling, solid and almost cartilaginous in consistency, about an inch long, and of the size of a blackbird's egg. It is movable, painless, and rarely tender. The skin is unaffected. The chin is a trifle rotated to the opposite side, and the head inclined towards the shoulder of the affected side. Out of 11 cases four were breech, and seven vertex presentation, but it is commonly stated



that three-fourths of these tumours occur in breech presentations, and only one-fourth in vertex presentations. It occurs in non-instrumental labour, especially if rapid, and must not be ascribed to undue violence. It is most frequent on the right side because of the common obstetrical positions.

As a result of twisting of the head or stretching of the neck during birth some of the muscle fibres are ruptured, and a certain amount of blood is effused. The tumour has been found full of pure blood (*hæmatoma*). More often there is little blood, and the swelling is the result of a primary or secondary myositis and proliferation of connective tissue. In a case of Volkmann's the whole muscle became fibrous, and of almost cartilaginous hardness. Possibly this was a syphilitic myositis, which chiefly affects the sterno-mastoid, biceps, and rectus abdominis muscles.

The tumour is slowly re-absorbed and disappears in 2 or 3 months, occasionally persists for 9 months, leaving no permanent deformity. Torticollis has ensued, a result more likely if the myositis is syphilitic. The diagnosis is easy, but the lump is sometimes mistaken for an enlarged gland.

In my experience no ill-effects have followed when no treatment has been adopted. Very gentle massage may be used twice a day. Blue ointment can be rubbed in. The muscle can be kept exercised and stretched in the opposite direction to prevent contraction. Too energetic measures may increase the myositis.

**Diaphragmatic Hernia.**—Over 500 cases of diaphragmatic hernia are on record, but it is rarely diagnosed during life. Eighty per cent. of 481 cases were on the left side (Paillard, 1903). Because of the mode of development of the diaphragm, which is completed in the second month of foetal life, they are situated in the posterior half and vary in type. In the *Embryonic* form, called Complete or Incomplete, according as the whole or part of the dorso-lateral element (Pillar of Uskow) of the diaphragm is absent, there is no sac, for it is developed before the complete closure of the pleuro-peritoneal septum. In the *Congenital* or *Foetal* type this septum is formed, but the muscular tissue is imperfect, and the hernia has a sac. An *Acquired* hernia is due to protrusion through an unusually large normal orifice, such as that for the œsophagus. The contents, in order of frequency, are stomach, transverse colon, omentum, small intestine, spleen, liver, pancreas, kidney, cæcum, and appendix.

Symptoms may be absent. Sometimes there is no characteristic cry at birth, and attempts at full inspiration are unsuccessful. Dyspnœa, cyanosis, recession of the lower ribs, and displacement of the heart may be present. Examination of the chest may reveal tympanitic resonance, varying with the distension of the gut and its contents; dulness due to the liver, collapsed lung, or fluid intestinal contents. Attacks of distressed breathing may be relieved by passage of flatus. Sometimes there are gastric symptoms and uncontrollable vomiting, due to intestinal obstruction. A female baby,



who died at the age of 5 months, presented in sequence attacks of dyspnoea and cyanosis, recession of the chest wall, and dulness over the right base ; and 2 hours later appeared in normal health with resonant note, though rather weak breathing, at the right base. Adenoids were removed under ethyl chloride without benefit to the breathing. The dulness varied from time to time ; recession was most marked on the right side ; and she died during an attack of collapse. The hernia included the large intestine, most of the small intestine, the left lobe of the liver, the gall bladder, and part of the pancreas. The liver was rotated on its axis, and extended to the umbilicus. The heart was not displaced. The middle and lower lobes of the right lung were unexpanded. In another girl, aged 10 months, apparently healthy till a fortnight before admission, vomiting and shortness of breath with displacement of the heart to the right, dulness over the left lower lobe behind, and absent breath sounds, were the chief symptoms. The breath sounds remained absent, but the note varied in resonance, sometimes hyper-resonant. The child suffered little distress, but died from cyanosis and respiratory failure. Both these cases were of the embryonic type.

In the diagnosis of this condition chief stress must be laid upon the variable character of the percussion note, absent breath sounds, displacement of the heart, respiratory difficulties, and vomiting or signs of intestinal obstruction. Some of the milder degrees of the congenital type are compatible with prolonged life. It is liable to be mistaken for pleural effusion, pneumothorax, and some form of pulmonary inflammation or collapse.



## CHAPTER VIII.

### ASPHYXIA NEONATORUM AND ATELECTASIS.

The stillborn babe is not necessarily dead, though it does not breathe or cry and may succumb. Lividity or Cyanosis is congenital or acquired. It may depend on ante-natal causes, the effects of labour, or post-natal factors. The maternal causes include death of the mother; interference with placental circulation from prolongation of the second stage of labour; mechanical compression or stretching of the cord; tetanus uteri, and asphyxial conditions. The foetal causes include damage to the respiratory centre by cerebral pressure or intracranial hæmorrhage; respiratory obstruction during and after birth; the entrance of secretions into the tubes and atelectasis; and various forms of congenital heart disease. The ante-natal signs of asphyxia are failing heart sounds and the passage of meconium. The usual appearances of asphyxia are found post mortem. The condition of the lungs depends on the mode of treatment and causation, e.g., great emphysema following on inflation, or abnormal secretions in the air passages.

Two varieties are described, though it is asserted by some that one of them is due to shock. In *Asphyxia Livida* muscular tonicity and cutaneous sensibility are present. The head, lower jaw, and limbs do not drop when the child is raised. There is general venous congestion; the skin is warm, and of a dusky or livid red colour, up to deep cyanosis; the mucous membranes are somewhat dusky. The pupils are normal; the tongue, lips, and face may be swollen; the heart beats slowly; the pulse is full, and the umbilical vessels are full of blood. Reflex excitability is retained, and the temperature is normal. Breathing may be absent, shallow and infrequent, or spasmodic; sometimes there are râles and hiccough. Generally the respiratory movements become more vigorous, and respiration is established. After being apparently normal it may slowly fail. The heart then becomes slower and weaker; the skin and mucous membranes of a livid pallor; and the condition passes into the second variety, and ends fatally.

*Asphyxia Pallida* is a further and more serious stage of asphyxia livida. There is marked loss of muscular tonicity; the head, lower jaw, and limbs drop when raised; and the whole of the body seems limp and relaxed. Anæmia is general and severe; the skin is pale or white and cold; the face of a livid pallor, and the lips blue. The eyes are glassy, and the pupils



more or less dilated ; cutaneous sensibility is slight or absent ; the reflexes are absent ; the sphincters are relaxed ; the temperature subnormal ; the heart impulse and sounds are weak, usually slow, and the umbilical vessels nearly empty. A few ineffective convulsive attempts at respiration may be made, but soon cease. Under energetic treatment breathing may be partially established, but it quickly fails ; the temperature falls and the end comes in a few hours or days. The air passages are often filled with mucus and amniotic fluid. Favourable cases pass through the stages of asphyxia livida before recovery.

Cases of mild or even severe asphyxia livida often recover under suitable treatment. The frequent association with intracranial hæmorrhage (*Apoplexy Neonatorum*), into the ventricles from the choroidal vessels or meningeal, makes the diagnosis and prognosis difficult. Hæmorrhage is generally recognised by the history of prolonged labour or the use of forceps, the presence of increasing coma or paralysis, and irregularity of respiration and failing pulse, in spite of treatment. It produces more or less mental impairment and spastic paraplegia. The outlook is still worse in asphyxia pallida. The unfavourable signs are dilated pupils, failing heart, and a fall of 3-4° F. It may be simulated by severe anæmia, due to profuse hæmorrhage from the cord. It is probable that mere asphyxia never leaves any permanent mental defect. If the lungs are not expanded death may ensue from atelectasis and secondary pneumonia.

*Treatment* must be directed to establishing or improving respiration and circulation, and maintaining the body temperature. The mouth and fauces are cleared of any mucus or secretion by swabs ; by a mucus evacuator, an india-rubber ball attached to a glass tube curved at one end ; by suction through a catheter ; or suspending the child by the heels. Respiration is stimulated by reflex irritation of the nose and fauces with a feather, or ammonia to the nostrils ; by dipping the child alternately in hot and cold water for a few moments, or douching with cold water after a hot plunge, finishing with heat ; by swinging the child in the air, or spanking the nates ; or by rhythmical traction of the tongue (Laborde) 8-12 times per minute ; or by artificial respiration. Schultze's swinging method consists of 6-8 successive swings, and then immersion in hot water 100° F., with friction and rapid rhythmical compression of the heart. Reflex irritation is valueless in asphyxia pallida. For this the cord is tied ; mucus removed from the air passages, while suspended head downwards ; and the child put in a hot bath, 110° F. and 4 ins. deep, in a supine position with a rolled towel under the head and shoulders, and a pair of clip forceps on the tongue and hanging out of the side of the mouth. Artificial respiration is adopted ; 2 oz. of hot saline injected into the rectum, and strychn. gr.  $\frac{1}{100}$  subcutaneously, and repeated in 15 minutes. At intervals the child is suspended by the legs for 15 secs. at a time, and the spine rubbed with whisky or liniment. On the whole simple rhythmical compression of the chest,



10 times a minute, is better than any other method of artificial respiration. It should be continued for a long time, until the heart stops, and as much as possible in a hot bath, avoiding undue roughness. In favourable cases the treatment must be continued until the lungs expand equally, otherwise secondary atelectasis results. Hot bottles, brandy, and oxygen are of assistance. Gasping inspiration can be assisted by elevation of the arms; and expiration in crying by compression of the chest. The signs of improvement are that the pupils begin to contract, the heart is stronger and more regular, the body less limp and flabby, the colour slowly darkens, and the reflexes return. Sometimes it is useful to inflate the lungs by mouth or catheter, with the nostrils closed. As the catheter is inserted into the larynx, not much force must be used. Most of the air returns by the side of the tube. In the mouth to mouth inflation the epiglottis may be forced over the glottis, fluids may be driven into the lungs, or air forced into the stomach. Skilled observation is necessary for 2 days as relapses are by no means uncommon, though breathing may have been going on satisfactorily for several hours.

**Atelectasis.**—Atelectasis, or imperfect expansion, is the name given to non-expansion of the lung, a persistence of the foetal state, found in the newborn and sometimes in older infants. The congenital variety is strictly the persistence of the foetal state, while the post-natal is really an acquired atelectasis or pulmonary collapse, due to mucus, etc.; getting into the bronchial tubes during labour. Congenital atelectasis is most frequent in the weak and premature, and is the chief cause of asphyxia neonatorum. A few cases are due to the pressure of diaphragmatic hernia; others have been ascribed to administration of ergot in prolonged labour, malformations and intra-uterine disease of the respiratory and circulatory organs; or to cerebral disease and hæmorrhagic pressure on the respiratory centre. The anatomical appearances are those of collapse with compensatory emphysema. It is more or less symmetrical, affecting the lower lobes and the posterior portions of the upper lobes, varying from a mere patch up to two-thirds of the whole lung. In a child, aged 31 days, both upper lobes were affected. The anterior edges, and often the superficial portions of the unexpanded areas, are emphysematous. The non-expanded lung is darkish red, solid, sinks in water, non-crepitant, vascular, and shows foetal lobulation. If the child has only lived a few days, the areas can be readily inflated. In prolonged cases secondary broncho-pneumonia and hypostatic congestion are often present. Hence arise difficulty in forcible expansion, and the need of microscopic examination for diagnosis. The right side of the heart is engorged and distended, leading to backward pressure, with hepatic and splenic congestion and enlargement. Often the foramen ovale is open, and the ductus arteriosus imperfectly closed.

At birth the child may be merely feeble or show temporary blueness, sometimes permanent. There is a history of difficult resuscitation or



asphyxia at birth, a feeble cry during the first few days, somnolence, and often a subnormal temperature. Physical signs are doubtful or absent. Cyanosis is out of proportion to the other symptoms, and is generally present in the cases which have not been completely resuscitated from the asphyxia. They gradually get worse, and die in a few days from asphyxia, asthenia, or convulsions. In others there is no cyanosis, yet the lungs are not completely expanded.

The infant remains feeble, sucks badly, cries feebly or not at all, progresses slowly or actually wastes, and has cold extremities and a low temperature. Cough and catarrhal sounds in the chest, paroxysms of dyspnoea, twitching, and lividity, general convulsions, and sudden cyanosis are sometimes present, or induced by slight causes, and last for 3 or 4 minutes. The prognosis must be guarded, for these attacks may be slight and easily recovered from, or at any moment severe and fatal from collapse or convulsions. Fatal syncope may occur quite suddenly. The duration and prospect of recovery vary with the extent of the mischief.

The *diagnosis* depends more on the history of asphyxia at birth, feebleness of the child and its circulation, the presence of cyanosis or temporary attacks of blueness, and malnutrition, than on physical signs. The latter are often slight or absent. If symmetrical and of a medium extent, it is impossible to be certain that the note is impaired, but a limited affection of one lung may give rise to differences in the physical signs on the two sides. As a rule the non-expanded areas are surrounded by over-expanded ones, and there is very little or no alteration in percussion. In prolonged cases dulness is found, perhaps limited to one base or apex, and the breath sounds are weak, and only occasionally somewhat bronchial. Attacks of cyanosis in newborns generally indicate atelectasis or congenital morbus cordis, and are often induced by over-distension of the stomach, by food or wind.

Secondary pneumonia gives rise to crepitations and fever.

*Treatment.*—Encourage crying in the newborn and in delicate infants; make them cry vigorously, at least twice a day for 10 minutes at a time, to induce lung expansion. Stimulating liniments to the chest, and a hot or mustard bath, twice a day for 5 minutes, are useful. Do not allow the child to lie much on its back; feed it on the breast or breast-milk by spoon; keep it in a temperature of 70-80° F., in an incubator if necessary. Sal volatile, brandy, and oxygen are necessary during the attacks of asphyxia.



## CHAPTER IX.

### THE UMBILICUS AND SEPTIC AFFECTIONS OF THE NEWBORN.

*The Cord at Birth—Abnormalities and Diseases of the Cord and Navel—Pyæmia Neonatorum—Buhl's Disease—Winckel's Disease—Erysipelas—Hæmorrhagic Disease—Suprarenal Apoplexy—Trismus or Tetanus.*

**The Cord at Birth.**—A few minutes after birth the cord is tied with sterilised tape  $\frac{1}{16}$ - $\frac{1}{8}$  in. wide, kept in saturated solution of boric acid, or with several strands of strong thread or silk, at a distance of 2 ins. from the navel and again a little beyond this. It must be tied sufficiently tightly to cut through Wharton's jelly. If not, the ligature may come loose as the jelly shrinks. The cord should be gently squeezed towards the navel before tying, to avoid the possible inclusion of a hernial protrusion, or Meckel's diverticulum, and strangulation. It is then cut between the two ligatures.

After the bath the cord is thoroughly dusted with boric acid or salicylic acid, 5 per cent. in starch powder, wrapped up in dry antiseptic gauze or wool, and fixed with strapping to the abdomen, the free end being directed upwards. Sometimes it is first disinfected with perchloride of mercury 0·1 per cent., carbolic acid 5 per cent., rectified spirit, or alcohol. It should be left alone and kept dry, until it undergoes desiccation or mummification in the next 3 days, and separation in 4-9 days, leaving a healed surface. The epidermis spreads from the periphery over the small raw surface, covered with secretion or crust, which may not heal until 2 or 3 weeks after birth. After separation of the cord an antiseptic dusting powder, and a pad of gauze are applied, and a broad piece of strapping or an abdominal belt is kept on for about a month to prevent umbilical protrusion.

**Abnormalities of the Cord.**—In the sixth to the tenth week of foetal life the cord contains loops of intestine, which are later withdrawn into the abdomen. The persistence of this condition is known as *Congenital Umbilical Hernia* (Hernia into the Umbilical Cord), or *Exomphalos*. This may rupture during labour, and the child be born with extruded viscera. It may be associated with other congenital defects, notably spina bifida. In two cases under my observation a large portion of the intestines protruded, covered by smooth transparent membrane, and forming a bluish



tumour. Other cases have contained both intestine and liver, ileum and Meckel's diverticulum, or the diverticulum alone. The tumour has a broad base or pedicle, and is fusiform or globular, the shape of half an egg or pear, as big as an apple, with the cord attached at the apex or lower portion. The covering membrane is sharply defined from the skin, and is composed of amnion and parietal peritoneum, which is transparent, or may be partially or entirely thickened. It dries up with the cord and separates. If small, the hernia may be withdrawn, and the umbilical area healed by granulation, or the exposed gut becomes covered by granulations, and the epidermis spreads over it. More usually it ends in general peritonitis, due to infection, and death. Of 32 cases operated on at birth 6 died; of 7 treated expectantly 4 died (Meyer 1905). Kindt's statistics; 50 out of 65 cured by operation.

*Amniotic Navel.*—The skin is deficient round the base of the cord and in the adjacent area, so that the amnion extends over this defective area and the cord. It dries, separates, and leaves a raw surface which heals by granulation.

*Skin Navel.*—The navel projects as a somewhat dome-shaped structure, due to the failure of the normal infolding of the skin at the edges when the cord separates. It gradually becomes normal as the child gets older. There may be hernial protrusion.

*Umbilical Hernia.*—The common hernia is a protrusion through a sharply-defined umbilical ring in the first months of life of a small piece of gut or omentum, occasionally a Meckel's diverticulum, from yielding of the umbilical cicatrix. It is associated with distension of the abdominal wall, and more or less separation (diastasis) of the recti. The peritoneal diverticulum, corresponding with the passage for the umbilical vein, predisposes. The exciting cause is straining set up by crying, vomiting, constipation, or cough. It is most common in girls and thin babies. The protrusion is button-like or resembles the short thick finger of a glove; the skin is normal or thinned by stretching; the navel scar is found at or below the apex. It may gurgle on reduction. During growth the protrusion is gradually retracted within the abdomen, and practically all get well before puberty, leaving no extra liability to hernia in later life. Treatment consists of careful diet, and an umbilical pad. A penny or similar disc is wrapped in lint and strapped on with wide, long strapping, applied so as to approximate the recti, and changed once a week; or an india-rubber umbilical belt can be worn. The pad should be large enough to well overlap the edges of the orifice, and worn for 3-12 months, or for 3 months after birth as a preventive. Operation is rarely required. If it is, the sutures should extend well above and below the navel, to approximate the recti. Occasionally strangulation occurs.

*Umbilical Fistula.*—The urachus, a remnant of the allantois, normally obliterated in the second month of foetal life, may be patent throughout



and discharge urine, giving rise to a congenital umbilical fistula. Neugebauer, of Warsaw, in 1890, collected over 40 cases. It is more common in boys than in girls, and due to urethral obstruction. It may form by eversion of the mucosa a small red tender tumour. It is treated by cauterisation or suture. Occasionally a cyst is formed in the median line, through closure of the duct at each end. Calculi have been found in a patent urachus. Another variety of fistula is due to patency of a Meckel's diverticulum, and is lined with columnar epithelium.

Non-congenital fistulæ may be umbilical or hypogastric. The former are usually due to abscess, communicating with the bowel and forming a fæcal fistula; the latter to wounds, abscess, or caries of the os pubis. The diagnosis depends on the character of the discharge.

*Umbilical Polypus.*—The simplest form is a *Granuloma*. This consists of exuberant granulations, forming a flat and sessile fleshy papillomatous, sometimes pedunculated, red tumour which bleeds readily, and discharges serum or sero-pus. It is found in the second or third week, perhaps hidden in the folds, and is due to the navel wound not healing. Untreated ones last for weeks but finally shrink. It is easily cured by nitrate of silver, powdered alum, or tannic acid. Ligature and cut off a pedunculated tumour.

The *Diverticulum Tumour* or polypus is covered by mucous membrane, like that of the small intestine, and is due to prolapse of a persistent Meckel's diverticulum.

In foetal life the intestine communicates with the navel by the vitelline or omphalo-mesenteric duct, leading to the yolk sac during the first 2 months of life. Normally this becomes obliterated towards the end of the seventh week of foetal life, leaving no trace. Super-involution may cause stenosis or atresia of the small intestine, e.g., imperforate ileum with umbilical fistula. The duct may persist as a ligamentous cord stretching from the navel to the gut; or a finger-like pouch communicating freely with the gut and attached by a cord to the navel, gut, mesentery, etc., or the cord free in the abdominal cavity. Bands of this nature are a common cause of intestinal obstruction under the age of 20. It frequently persists as a simple blind pouch a few inches long. This may become constricted and not communicate with the gut; it may then dilate into a cyst which discharges mucus, if the rest of the duct is pervious and opens at the navel. Sometimes it persists as a pervious duct throughout, discharging fæces at the navel. A fæcal fistula is occasionally due to the projection of a persistent diverticulum into the umbilical cord, and inclusion in the ligature applied at birth. A pervious duct gives rise to a simple umbilical fistula, or, by eversion of the mucosa, to a red raspberry-like polypus, from a pea to a cherry in size and sometimes much larger, through the centre of which a probe can be passed into the duct. If the prolapse is extreme two lateral fistulæ are produced. There is a history of navel discharge, intestinal in



character. If there is no polypus, the duct may be closed spontaneously, by applying a ligature, or on cauterisation. Usually it is necessary to excise the diverticulum in order to cure the fistula and prevent possible intestinal strangulation.

Meckel's diverticulum is present in 1-2 per cent. of all people. It is situated between the duodenum and cæcum, usually at right angles to the duct, at a distance of from 10-80 cm. above the ileo-cæcal valve, the distance increasing with the age of the patient. It may have a definite mesentery. It is a cause of umbilical fistula and polypus, enterocysts, intussusception from inversion, intestinal obstruction, torsion, volvulus, and strangulation. It may undergo inflammation, ulceration, and perforation, and simulate appendicitis. Indrawing of the umbilicus or excess of umbilical scarring indicates adhesion to the abdominal wall or past fistula.

*Umbilical Eczema*, acute or chronic, is due to sebum and dirt collecting in the sulci. It extends to the summit of the folds and adjacent skin, and gives rise to œdema, exudation, itching, crusts, and bleeding. Apply a powder of calomel, zinc oxide, and starch.

*Umbilical Hæmorrhage*.—Bleeding from the cord or navel results from accident or disease. *Accidental Hæmorrhage* takes place from the larger vessels, and is due to inefficient ligation of the cord, the ligature too narrow and too tight, too loose, or tied too near the navel. The arteries ought to contract, without ligation, sufficiently to prevent bleeding. Failure to do so depends on prematurity, asphyxia, morbus cordis, and other causes of high blood pressure. Then, on shrinking of the cord, the ligature becomes loose and bleeding occurs. A second ligature must be applied. If the cord is too short or has dropped off, pass a hare-lip pin for about 1 in. through the abdominal wall at the lower level of the umbilicus, and apply a figure of eight loop. Two pins at right angles to each other may be needed. If this fails to stop the bleeding, do an abdominal section, and ligature the cord before exit. If the bleeding takes place from the navel, it may be due to forcible avulsion of the cord, ruptures or tears, or excessive granulations. *Spontaneous Hæmorrhage* from cord or navel is due to sepsis, hæmophilia, or congenital syphilis; it usually takes the form of capillary oozing. Sometimes it is sudden and profuse. It begins after separation of the cord and occasionally takes place into the cord, before it has dropped off, within 24-48 hours of birth. It may occur alone or with other hæmorrhages. For treatment *vide* Hæmorrhagic Disease.

**Sepsis or Pyæmia Neonatorum**.—Infective organisms reach the foetus in utero through the maternal blood. During birth infected liquor amnii or secretions may be swallowed or aspirated into the air passages. After birth the infecting agent is conveyed by dirty hands, ligatures, powders, dressings, bath water, flannels or sponges, instruments, clothes, utensils, etc. The site of infection is commonly the umbilical cord or navel, viâ the



blood vessels or lymphatics. Less often the infection enters through an abrasion of the skin; mucous membrane of mouth, fauces, naso-pharynx, or conjunctiva; respiratory or alimentary tract; or the genitals. Sometimes the source of infection cannot be found or is overlooked. Thus, the pyæmic origin of severe broncho-pneumonia may not be recognised unless the cord, navel, etc., are examined. In one baby the infection was secondary to a general mild pustular dermatitis; in another, to a patch of gangrene in front of the ear, due to the pressure of forceps during delivery. Four-fifths of the cases are umbilical in origin. Bottle-fed infants are more susceptible than the breast-fed for they lack the protective antigens of the colostrum. The common infective organisms are streptococci and staphylococci. Premature, young, and weak infants are very prone to septic infection because of their low resisting power, undeveloped glandular system, and late closure of the umbilical vessels.

*Infections of the Cord.*—Mild forms of infection lead to putrefaction and delay in separation of the cord. Rarely the cord undergoes a kind of organisation, and forms a tumour which requires removal. More severe infection causes moist gangrene or sloughing, with softening, offensive odour and discharge. Anomalies in separation of the cord, and in healing of the navel after separation, and umbilical infections are most liable to occur in weak and premature infants. Separation of the cord must never be hurried by mechanical means, or a raw surface and liability to hernia are produced. For infective conditions powder with iodoform after cleaning with 1 in 5000 perchloride lotion.

The navel heals quickly under normal conditions, but is a frequent source of infective disease. Among the chief affections may be mentioned omphalitis, gangrenous lymphangitis, umbilical ulcer and gangrene, subnavel abscess, arteritis and phlebitis, and general infective conditions, including enteritis, pyæmia and general sepsis, hæmorrhagic disease, Winckel's disease, Buhl's disease, erysipelas, and tetanus.

*Omphalitis* is an inflammation of the skin and cellular tissues of the navel, with redness, swelling and sero-purulent or purulent, sometimes hæmorrhagic discharge, and the formation of granulations. These may be profuse and polypoid (*v.* polypus) in appearance. A milder type may be due to chemical and mechanical irritants. In the infective form the dusky inflamed ring at the navel spreads peripherally and may reach the pubes. The abdominal wall becomes brawny and œdematous. Its onset is in the second week before the navel has healed. It gives rise to fever, anorexia, whining, abdominal distension, and general malaise. The course varies with the severity of the infection; it may become superficially very extensive. It may undergo resolution, terminate in one or more abscesses, ulceration, gangrene of cord or navel, or sloughing and secondary peritonitis.

*Gangrenous Lymphangitis* is commonly the result of umbilical infection and affects the umbilicus, scrotum, thighs and crural arch. It begins



with the red striæ of lymphangitis, œdema, and slight general redness. The public region, scrotum, and penis may become suddenly red and swollen. The scrotum gets hard and tender, dark red, with purplish patches and occasionally bullæ on the surface. It may become necrosed, exposing the testes. During healing these bodies may be included in scar tissue and subsequently atrophy. It simulates erysipelas, gives rise to signs of general infection, and is treated by free incision with the thermocautery.

*Umbilical Ulcer and Gangrene.*—An ulcer is situated at the bottom of the navel and often hidden by folds. It is usually from  $\frac{1}{4}$ - $\frac{1}{3}$  in. in diameter, but may extend to the skin and become large. The stump of the umbilical artery may be visible. In gangrene a dirty scab forms at the umbilicus, and is surrounded by a ring of dusky infiltration which spreads outwards and inwards. Bullæ sometimes form. It occurs in feeble and ill-nourished babies, usually secondary to omphalitis. It causes destruction of the muscles, peritonitis, perhaps extrusion of viscera, fæcal fistula, or severe hæmorrhage. It generally ends fatally, in collapse or coma.

*Subnavel abscess* occurs in the course of the vessels, or is secondary to and beneath a localised omphalitis. Redness is commonly absent, but there may be some local tenderness, just under the navel. Sometimes there are no local symptoms, and it is only found after death. It may discharge through the navel, spread by the lymph channels to the peritoneum or general system, and very occasionally rupture into the peritoneal cavity. All these conditions are treated on ordinary surgical principles.

*Arteritis and Phlebitis.*—Arteritis is much more common than phlebitis, and is due to extension from the connective tissues of the cord, or secondary to local ulceration or omphalitis. Usually it is a peri-arteritis, not an arteritis. It may be limited to the cord or extend as far as the bladder. The vessels contain septic thrombi and pus which can sometimes be squeezed out from the end of the cord. It may lead to thickening of the arteries, palpable under the abdominal wall, and to abscesses between the peritoneum and the abdominal wall. If the cord has separated, there may be no visible changes at the navel, though the mischief is great and toxæmia profound. Occasionally it gives rise to general pyæmia, involving the viscera, joints, and bones, but not producing jaundice. It lasts from days to weeks, and is fatal from sepsis, peritonitis, pneumonia, or malnutrition.

Phlebitis on the other hand rarely gives rise to local signs, unless it is associated with arteritis. It affects the umbilical vein, and causes thrombosis, pus formation, and secondary hepatitis, with jaundice which increases daily and becomes intense. Pus can rarely be squeezed out of the vein. Abscess may be found just beneath the navel, anywhere in the course of the vein, or under the liver. General pyæmia ensues. The liver shows acute interstitial and parenchymatous inflammation, and often multiple abscesses. Portal thrombosis, peritonitis, and pneumonic affections of the lung are not



infrequent complications ; umbilical gangrene may occur, and occasionally endocarditis. The spleen is enlarged and soft ; fatty changes are found in the liver, spleen, and kidneys, and embolic foci in the kidneys. Phlebitis is practically always fatal. Comparatively, the prognosis of arteritis is favourable, though very bad, for it may be quite local.

*Pyæmia.*—From the above description there are clearly two types of pyæmia in the newborn. One corresponds in character with pyæmic infection generally, the other is distinguished by its effects on the liver and jaundice. Both may result from infection through the cord or navel, and the disease may be local or general. If due to infection through the cord it begins 2-5 days after birth. If due to umbilical infection it may occur at any date before the navel has healed. The onset is characterised by fever, gastro-intestinal derangement, loss of weight, and erythematous rashes, and by early jaundice if due to phlebitis.

Various types may be described. It may give rise to no symptoms except sudden collapse, rapid fall of temperature, and death ; this type is most common in the premature. Often it simulates acute gastro-enteritis, with vomiting, diarrhœa, and high fever. Occasionally there are severe cerebral or meningeal symptoms. Thus, a child developed a pustular rash on the fifth day of life and twitching on the seventh day ; next day the temperature rose to 105° F., fever persisted for 3 weeks with almost constant attacks of general tonic and clonic spasms, in which the child sometimes became livid. The spasms ceased entirely 2 weeks after the fever subsided, and apparently left no permanent affection of the brain. They were probably due to toxæmia. Or the infection assumes a pneumonic type ; or is indicated by fever, greyish pallor, and hæmorrhages, e.g., many cases of melæna.

*General Symptoms.*—Fever usual at the onset, variable in its course, and subnormal temperature at the end ; it may be very high, even up to 109° F. Restlessness, crying, and disturbed sleep, tremors, twitchings, tetanic spasms of the muscles, a torpid or comatose condition, and rarely convulsions. Jaundice, cyanosis, œdema of the feet, and sclerema, especially in the premature. Erythematous rashes, petechial hæmorrhages, purpura and bullæ ; epistaxis, hæmaturia, hæmorrhages from mucous membrane, retinal hæmorrhages. Furunculosis, skin abscesses, and bed sores. Rhinitis, stomatitis and otitis. Dyspnœa, bronchitis, pleurisy, broncho-pneumonia, empyema, and infarction of the lung. Gastro-enteritis. Vomiting is not common. The stools are green, undigested, often offensive, and may contain blood. Diarrhœa is common. Encephalitis, septic meningitis, purulent pericarditis, purulent bronchitis, minute abscesses in the lungs, purulent arthritis of one or several joints, and subcutaneous abscesses are occasional complications.

The blood is deficient in red cells, in coagulability, and may contain an excess of polymorphs. The red cells undergo destruction, and iron



pigment is deposited in the tissues. Sepsis produces acute cellular degeneration or undue fibrosis of the liver, functional disorders, often jaundice; changes in the vessel walls and congestion of organs, inducing hæmorrhage.

*Post mortem* appearances include parenchymatous degeneration of the heart, liver and kidneys; hæmorrhages into the skin, under the pleura, on the surface of the brain, and in one or more of the other organs. Encephalitis and meningitis are occasionally found.

The *diagnosis* is usually comparatively simple. Fever and jaundice, apart from local evidence, are very suggestive in those conditions in which infection is liable to occur. Peritonitis is a common complication; indicated by tympanites, tenderness, effusion, vomiting, flexion of the thighs, and protrusion of the umbilicus. It may be localised about the umbilicus or liver, but is more often of the general septic type seen in older patients. Diarrhœa and broncho-pneumonia may be the only signs; the latter gives rise to rapid breathing, cyanosis, and recession, but the physical signs may be of little assistance. Cases have to be diagnosed from acute gastro-enteritis, pneumonia, atelectasis, and meningeal hæmorrhage. Bacteriological examination of the blood is inefficient for there are few organisms present, and it is difficult to obtain enough blood for thorough examination.

The *prognosis* is worst in the weak and premature, and depends on the extent and severity of the infection, and the nature of the infective organism. Erysipelas and tetanus are almost always fatal. Cases in which the liver, peritoneum, lungs, or pleura are involved nearly always die. Pyæmia is often fatal in 2-5 days. Its course is very variable, and symptoms may be slight or absent. Loss of weight is rapid, and may be the only symptom. A sudden increase shortly before death may be due to œdema or ascites. Hæmorrhages are common and may be fatal. Death is sometimes quite sudden.

**Buhl's Disease** begins on the fifth or sixth day of life, and has been ascribed to asphyxia at birth. The child may be cyanotic from birth, or the lividity may subside, but breathing remain weak. It gradually becomes feeble, develops swelling of the subcutaneous tissues, pitting of the skin, severe anæmia, jaundice, multiple hæmorrhages, and dies from collapse in the course of 2 weeks. Acute fatty degeneration and parenchymatous inflammation are found in the heart, liver and kidneys, the cells of the pulmonary alveoli, and muscles; much as in acute yellow atrophy and phosphorus poisoning. Probably it is a variety of sepsis.

**Winckel's Disease** is characterised by jaundice, cyanosis, and hæmoglobinuria. It is sometimes called epidemic hæmoglobinuria, icteric cyanosis, *maladie bronzée*, or hæmaturic bronzed disease. It is very rare. Charrin (1873) reported 14 cases. Bigelow observed it in epidemic form in 1875; and Winckel, in 1879, fully described 23 cases in a Dresden Hospital. It has generally occurred epidemically in institutions, and is probably due to some infection. The red blood cells undergo rapid disintegration.



It begins during the first week of life, generally on the second day, and proves fatal in a day or two from rapid asthenia, convulsions, or coma. The symptoms are restlessness, whining, anorexia, vomiting or bilious diarrhœa, frequent pulse and respiration, cyanosis, jaundice, and sometimes slight convulsions. The bronze colour of the skin, which may become as dark as that of a mulatto, is due to the combination of jaundice and cyanosis. Fever is slight or absent, and prostration profound. The urine is passed often, in small quantities, with pain and straining. It is brown or smoky, contains hæmoglobin, and sometimes renal epithelium, granular casts and blood cells, and occasionally albumin.

After death there are found fatty degeneration of the organs; many hæmorrhages, especially in mucous and serous membranes; a large spleen filled with masses of granular blood pigment; enlarged liver; large kidneys containing hæmorrhages, and altered urine in the bladder. The umbilical vessels are usually normal. Possibly there is a hæmaturia, not a hæmoglobinuria.

**Erysipelas** is generally umbilical in origin, coming on at the end of the first or beginning of the second week. If due to infection of the cord it begins earlier. It may cause a mild local induration, or a general infection with severe local and general symptoms. The local redness and œdema have a well defined border, and spread rapidly downwards over the lower abdomen and limbs, rarely upwards, occasionally over the whole body. Sometimes bullæ form, and often superficial areas of necrosis. It may cause sloughing of skin and exposure of muscles. Fever may be absent or high. Vomiting, diarrhœa, drowsiness, and fits occur. It spreads rapidly, and death results from asthenia or peritonitis. Bouchet regards it as invariably fatal in the newborn. Of 30 cases under one year of age 16 died (Billard). After the first fortnight of life it may be due to infection through microscopic abrasions or cutaneous inflammations, e.g., in napkin area. A few cases are secondary to vaccination. Treat on general principles; maintain the strength as far as possible; and apply locally peroxide of hydrogen, iodoform, or ichthyol, 10 per cent. in lanoline.

*General Treatment of Sepsis Neonatorum.*—Prophylaxis is of the utmost importance, for in quite three-fourths of the cases infection is umbilical and preventable. The disease is the fault of some one or other of the attendants. The local condition is treated on ordinary surgical principles. Nervous symptoms are relieved by 6 hourly baths at 90-95° F. for 5-10 minutes, and 4 hourly small doses of phenazone or phenacetin. Spasmodic conditions are best relieved by chloral, with or without bromide, by mouth or rectum. Caffein, digitalis nux vomica, brandy, sal volatile, and saline injections are valuable in collapse and debilitated conditions. Keep the child warm, and be sure that the diet is simple and easily digested.

**The Hæmorrhagic Disease of the Newborn.**—Hæmorrhage in the newborn may be *Accidental* (p. 122); *Traumatic* from injury during labour



(p. 108); or *Pyogenic*. The last variety is sometimes called *Hæmatemesis* or *Melæna Neonatorum*, but these terms include also rare cases not due to sepsis. In true Hæmorrhagic Disease there is a spontaneous capillary oozing, which begins at a variable period after birth, and may prove rapidly fatal, or last for a few days to a few weeks, and yet end in recovery. It occurs in the well nourished as well as in the weakly; is more frequent in males than females; and may be epidemic in institutions. The bleeding takes place from the umbilicus; from the mucous membrane of the nose, mouth, stomach, or intestines; from the genito-urinary organs; from any abrasion of the skin or subcutaneously; into the thymus, lungs, and various abdominal viscera; into the serous cavities; intracranially; and even through the skin of the palms and soles.

Predisposing factors include prematurity, congenital debility, and bad hygiene. The circulatory disturbances, due to ligature of the cord at birth or compression of the cord during labour, may cause hyperæmia of the mucous membranes and throughout the body, hæmorrhagic oozing, and extravasation. Similar congestion can be produced by backward pressure from congenital heart lesions, the respiratory obstruction in asphyxia and atelectasis, and hepatic cirrhosis. Gastro-intestinal hæmorrhage may thus be a kind of epistaxis from the congested membrane in consequence of its sudden functional activity, possibly associated with deficient coagulability of the blood. Or the congestion and hæmorrhage may be set up by purgatives and ingesta. Sometimes the bleeding comes from a duodenal or gastric ulcer, still more rarely an œsophageal one. Care must be taken to exclude hæmatemesis and melæna due to blood swallowed during parturition or suckling from a fissured nipple, epistaxis, wounds, ulcers, and sores of mouth and gums. Congenital syphilis is regarded as an important predisposing factor. Abt, of Chicago (1903), found it present in 2 out of 12 cases, but Machell, of Toronto, obtained no evidence of it in either child or parent, in 14 cases. Undoubtedly hæmorrhage can occur in congenital syphilis. Endarteritis of small vessels, possibly syphilitic, has been found in some fatal cases. The worst cases which have come under my notice have not been associated with any trace of this disease. The affection is not due to hæmophilia, for hæmophiliacs rarely bleed before the end of the first year, and infants who recover from hæmorrhagic disease do not subsequently prove bleeders.

The important attacks are those due to pyogenic infection by various organisms, present in many cases and giving rise to pyæmia or toxæmia. Possibly toxins reach the foetus in utero, traversing the placenta; or the infection is a sepsis neonatorum. On the other hand sepsis and hæmorrhage often occur independently of each other. Some change is produced in the blood or blood vessels, permitting extravasation. The effects depend on the amount of blood lost and the causation.

*Gastro-intestinal bleeding* may be taken as the type of this disease. As



a rule hæmatemesis starts from 12-36 hours after birth. It may begin at the sixth hour, be delayed until the sixth day, or even as late as the sixth week; varying with its site and causation. Few cases begin after the first week. Bleeding is the first sign. In one instance fever was present from birth, melæna began 12 hours and hæmatemesis 18 hours after birth, lasting for 12 hours more and ending in recovery. Another apparently healthy child passed bright red blood on the seventh day, got rapidly worse with more severe bleeding, and died on the tenth day; there was no jaundice or fever. A third case was that of a premature baby, under 4 lbs. in weight, with a little jaundice, but no fever; melæna began on the seventh day, and ended fatally on the tenth day, no cause being found post mortem. In other instances the cases were of the type described as sepsis in the newborn, coming on with fever a few days after birth, the hæmorrhage being a subsidiary symptom. The blood may be dark in colour at first and contain clots, if it comes from the stomach or high up in the intestine. In profuse hæmatemesis or bleeding from the colon it is bright red, thin, and watery. Bleeding may begin before food has been taken, or follow vomiting or the passage of mucus. Meconium is evacuated first, next dark or very tarry blood mixed with fæces, and perhaps finally profuse bright red watery blood. Bleeding may take place also from the nose and navel, from any single site, or from several places and into various organs more or less simultaneously. Melæna and hæmatemesis occur in one-fourth of all hæmorrhagic cases, of which 50-60 per cent. are melæna only; 15-20 per cent. hæmatemesis without melæna. Both are often associated with bleeding from other sources, jaundice, and fever.

*Diagnosis.*—The diagnosis is important in view of the serious nature of the true hæmorrhagic disease. Care must be taken to exclude hæmatemesis due to swallowed blood. Gastro-intestinal hæmorrhage may be concealed until melæna is noted, or even prove fatal before blood appears in the stools. Hæmaturia may be due to uric acid infarcts; vaginal hæmorrhage to local congestion, injury, or polypus; hæmorrhage from the ears, due to fracture of the base of the skull at birth; bleeding from the eyes or into the retina, due to injury. Generally the diagnosis is based on the multiplicity of the hæmorrhages, the date of onset, and the presence of fever. Fever may be absent. In mild cases the bleeding is slight, and ceases in a few hours or a day or two. Some are more prolonged and serious, with loss of weight, anæmia, and prostration. The greater and more sudden the bleeding, the worse is the prognosis. Umbilical and vaginal hæmorrhage are the least serious. Gastro-intestinal hæmorrhage, with little or no fever, is not grave unless the bleeding is profuse, bright red, and watery. One or two attacks of severe hæmatemesis may be recovered from. A purpuric rash is a very bad sign. If only the septic cases are regarded as the true hæmorrhagic disease the prognosis is very bad. It is bad if the blood is bright red and continuously oozing from a mucous membrane; if high



fever and jaundice are present, indicating sepsis ; if there is an enlarged spleen or history of syphilis ; or if there is diarrhœa, common in septic cases.

Mortality statistics vary from 35-78 per cent., according to the type of cases included. In fatal cases death results from profound anæmia, collapse, asthenia or convulsions. Death or recovery ensues in 3 or 4 days. Usually nothing abnormal is found post mortem ; sometimes acute fatty degeneration, umbilical and portal phlebitis, enlargement of spleen and liver, or syphilis.

For local bleeding apply calcium salts, adrenalin, or sterile gauze soaked in gelatine solution, 10 per cent. Accidental bleeding is treated by astringent powders, alum and tannic acid, or cauterisation. In all forms of the hæmorrhagic disease give gelatine, with water or whey, by the mouth ; a 2-5 per cent. solution by the rectum ; or in very severe cases a 2 per cent. solution in normal saline in doses of 10-15 c.c., every 3-6 hours subcutaneously. The fluid must be boiled for 6 hours first, as it may contain tetanus organisms. Sometimes it gives rise to toxic symptoms. In addition, give hazeline, hamamelis, calcium lactate, and bismuth by the mouth for bleeding from the alimentary tract. Avoid irritating food and keep the child warm.

**Suprarenal Hæmorrhage or Apoplexy** may be traumatic, the result of injury during labour. Usually the child is stillborn, death occurring before or during labour. Undoubtedly the delicate, fragile character of the circulation in these glands is a predisposing factor. The vessels may give way in consequence of asphyxial conditions or compression of the cord. In some instances congenital syphilis or hæmophilia may be to blame. Rare causes include convulsions, vaso-motor disturbance, and thrombosis of the inferior vena cava or renal vein. Probably the most constant cause is some form of septic infection. The amount of hæmorrhage varies from severe congestion to the production of a large bloody tumour, the size of an orange. It is often bilateral. In the newborn it is frequently associated with other hæmorrhages ; and in older children with purpuric rashes and high fever, sometimes due to cerebro-spinal fever. The onset is sudden, perhaps with screaming, acute abdominal pain, vomiting and convulsions. The child soon becomes seriously ill, the temperature rises, diarrhœa is present, and purpura is not uncommon. Death may take place in a few hours, or in a few days, from collapse, or with symptoms like peritonitis. Riviere (1902) collected 6 cases of this type, and added 4 more. In his patients the Peyer's patches and mesenteric glands were enlarged. Bacteriological examinations proved negative. He suggested intestinal toxæmia as a possible explanation. In a child, aged 3 months, with somewhat similar symptoms, viz., anorexia, convulsive movements, drowsiness, and death from collapse, 53 hours after the onset, I found (1908) an extensive bilateral renal apoplexy of uncertain causation.

**Trismus or Tetanus.**—Trismus or Tetanus of the newborn, sometimes known as the "Eight Days Sickness of St. Kilda," is due to the tetanus



bacillus infecting the umbilical wound, or rarely, viâ the skin or genitals. It is common in the tropics, especially the West Indies, Cuba, and America; and comparatively rare in Europe. In 1782 one out of every six babies born in the Rotunda Hospital, Dublin, died from it. At St. Kilda, in 1880, 14 couples had had 125 children, of whom 84 died from trismus during the first 14 days of life. The organism is widely distributed in the dust of streets and houses, and is conveyed by the hands of the attendant or the bandages, pads, charred brown paper, Fuller's Earth, or ointment applied after birth.

Trismus commonly begins on the fourth day, and rarely later than the tenth day after birth unless the navel is unhealed. The first sign is difficulty in suckling, and apparent anorexia because of the trismus or spasm of the masseters on suckling. There are marked restlessness; frequent sharp cries; general corrugation of the forehead, facial muscles, and lips; the "risus sardonicus"; retraction of the head and opisthotonos. The limbs are rigid, the upper ones acutely flexed, and the lower ones usually extended. The pulse and breathing are increased in frequency. The temperature rises to 104-106° F. or more, and often there is a further rise after death. Sometimes there is no fever. Gradually sucking and swallowing becomes impossible, the spasms or fits are more and more frequent, or longer in duration, induced by the slightest external stimulus, and ending in cyanosis. The breathing becomes shallow and irregular, and death ensues from asthenia, spasm of the larynx or spasm of the diaphragm. The navel may appear normal, or discharge pus. Although the organism can grow in pus, pure cultures are rapidly destroyed at the seat of inoculation by the toxin produced. Early diagnosis is important. The spasms and rigidity must be distinguished from those due to developmental errors of the cerebrum, encephalitis, meningitis, and cerebral toxæmia.

Some cases are quite mild, and even limited to trismus. A few continue many weeks and recover. These chronic cases generally come on a few days later in life with twitching, jaundice, and a peculiar pallor. Of the acute cases many die within 48 hours of the onset. Finlay, of Havana, states that 131 out of 141 died in the first week and 10 in the second week of life.

Tetanus in older children is common in Australia, and in the United States of America about Independence Day, July 4th, due to wounds by the blank cartridge of toy pistols. The earliest symptoms are temporary local contractions at the site of the wound, sighing and yawning, or other signs of dyspnœa up to cyanosis and glottic spasm. Stiffness of the neck, jaw, back, and abdomen develop 1-7 days later; constipation is very common. Its course is similar to that in adults. In both babies and children the prognosis depends upon the duration of incubation, the nature of the wound, the height of the temperature, and the duration of the symptoms. The incubation period averages 11 days; less in the newborn. If only 10 days, 4 per cent. recover; if 11-15 days, 27 per cent. recover (Rose).



After a duration of 5 or 6 days recovery is probable. The outlook is less serious in the so-called idiopathic cases, and worse in those secondary to abrasions and jagged irregular wounds. It is favourable if the temperature is under  $101^{\circ}$  F., and very serious if over  $103^{\circ}$  F.

Local treatment consists in the use of antiseptics; cauterisation; incision and curetting, swabbing with pure carbolic, and packing with iodoform gauze; excision or amputation. Babies should be fed on the breast-milk, given by gavage if unable to swallow. Protect them from external irritants, cold, draught, noise, etc., and give a dose of calomel or an enema, and a warm bath every 3 hours. Give chloral in doses of grs. 1-5, every 2-4 hours, combined with double the quantity of potassium bromide. Continue this from 24-48 hours, and then in smaller doses. Atropin gr.  $\frac{1}{8000}$  -  $\frac{1}{3000}$ , curare gr.  $\frac{1}{50}$ , physostigmin gr.  $\frac{1}{100}$ , and phenol have been given 3 times a day subcutaneously. For high temperature the coal tar products are better than hydrotherapeutic measures. Chloroform inhalations relieve the spasms temporarily. Anti-tetanic serum can be tried, but has not proved of much value. It neutralises toxin formed at the seat of inoculation and circulating in the blood, but does not affect it when it has combined with the nerve cells. It may cause rash and painful synovitis. It may be injected in the neighbourhood of the wound, intramuscularly into the buttock, intravenously, or by lumbar puncture. Behring's and Tizzoni's serums are the best. Meltzer found that magnesium salts inhibited the functional activity of the nervous tissues, and caused surgical anæsthesia in monkeys on lumbar injection. It may be injected in doses of 1 c.c. of a 25 per cent. solution to every 25 lbs. of body weight every 36 hours for 4 doses, and sometimes controls the rigidity and convulsions. It is very difficult to estimate the value of remedies, because of the impossibility of judging accurately the course of the disease, if untreated. On the whole it is best to rely upon chloral, careful feeding, nursing, and warm baths. Morphia prevents elimination and may do harm.



## CHAPTER X.

### JAUNDICE.

*Icterus Embryonum*—*Icterus Neonatorum*—*Biliary Concretions*—*Pyæmic Jaundice*—*Congenital Defects of the Bile Ducts*.

In rare instances the infants of women suffering with prolonged jaundice have been born similarly affected. Still more rarely the mother is apparently healthy. This is known as *Icterus Embryonum*. These babies may be premature, stillborn at full time, or may live. Jaundice at birth is occasionally seen as the result of syphilitic hepatitis, malformations of the bile ducts, or in the rare cases of family acholuric jaundice.

**Icterus Neonatorum** is a common form of jaundice in newborns, varying from a pale to an intense yellowness. It appears usually on the third to the fifth day of life, rarely on the first, and not often after the fifth; and is found in from 30-85 per cent. of all babies, more especially in the premature, the small, and the firstborn. It is first noticed in the nose and cheeks, and may be limited to the face. Next it affects the thorax, abdomen, conjunctivæ, and extremities. The sclerotic is not always involved. By pressure of the finger on the skin a yellowish print is produced. In the early red or pre-icteric stage the yellowness is partly concealed by the hyperæmia of the skin. The yellowness increases in depth for two or three days, and then slowly disappears. In mild cases the urine is unaffected, but in more severe ones it may contain bile salts, and pigment. Golden yellow, shining, insoluble masses of pigment have been found in the urine. They are soluble in alcohol, soluble in strong  $\text{H}_2\text{SO}_4$  with red colouration, and insoluble in ether. These are masses of bilirubin (Cruse). Bilirubin is feebly soluble in the urine of the newborn, because of the scanty amount of alkaline phosphates. The colour of the stools is unaffected, except in very bad cases, and nutrition rarely suffers. It lasts for a week or two, and occasionally for many months, without making the child ill, and ending in complete recovery. In prolonged cases the occasional presence of bile in the stools is a favourable sign. A fatal issue may ensue on some coincident affection, and post mortem every organ in the body is more or less bile-stained, except the liver, which is rarely discoloured, or only in patches. Bilirubin crystals, the "hæmatoidin infarcts" of Virchow, have been found in the apices of the renal papillæ, and are peculiar.



Its pathology is uncertain. At birth the abrupt cessation of the foetal circulation induces more or less congestion of the liver and venous stasis. The dilated veins press on the bile capillaries and ducts, and cause retention of bile in the liver. This condition has been demonstrated in fatal cases, and is favoured by anything which interferes with the expansion of the lungs, and the action of the heart; hence jaundice is more common in feeble infants and in those born asphyxiated.

It has been ascribed to the rapid destruction of the red cells in the first few days of life, and solution of the colouring matter in the serum which reaches the tissues, where it is reduced to some kind of yellow pigment. It is no longer generally accepted that any such disintegration of red cells takes place after birth. Estimations based on the number per c.mm. are unreliable for the percentage varies with the quantity of plasma. Moreover, the occasional presence of bile acids in the urine indicates that the affection is not hæmatogenous.

In all probability an increased formation of bile takes place at birth, due to the stimulus of a rich blood supply (Knöpfelmacher), and the capillaries become overdistended with viscid tenacious secretion at a low pressure, in consequence of which some of it passes into the blood. It may therefore be regarded as a physiological manifestation. It is unlikely that it can be due to any more serious cause.

No special treatment is needed, but small doses of calomel or grey powder, and bicarbonate of soda may be given. In making a diagnosis we have to exclude those cases of jaundice due to septic infections, Buhl's disease, and Winckel's disease, syphilitic hepatitis, malformations of the bile ducts, and the rare cases of family acholuric jaundice. A tendency to the secretion of viscid bile may be a family peculiarity in those cases where many successive infants have died from jaundice in the first month of life. It is important to remember that simple jaundice in the newborn may be very prolonged, and yet not necessarily due to malformation of the bile ducts, sepsis, or liver disease. Nevertheless, if the discolouration increases in intensity in the second week, it is in favour of a more serious diagnosis. Severe jaundice from any cause may lead to multiple hæmorrhages.

**Biliary Concretions** are more common in the liver of infants than at any other period of childhood, and even occur in the foetus. This is a further proof of the tendency to stagnation of bile at this age. Intense jaundice at birth or shortly after results, and calculi are found in the ducts in fatal cases. Stone in the gall bladder is extremely rare before puberty, and gives rise to the usual symptoms of colic and jaundice, and the passage of a stone. In a boy of 4, who had attacks of colic for 2 years, the gall bladder was dilated, and the cystic duct contained a stone (Friedlander, 1907).

*The Gall Bladder* is very rarely absent. It may be transposed, bifid or duplicated, or show hour-glass constriction. Dilatation is generally due to obstruction, but may occur without any evidence thereof.



**Malformations of the Bile Ducts** are rare. The common duct may be absent, represented by a fibrous cord, or obliterated at its entrance into the duodenum. The cystic and hepatic ducts, alone or associated with some defect of the common duct, may be impervious. Developmental errors and obstruction are ascribed to adhesions from foetal peritonitis, foetal cholangitis, and congenital syphilis. If the cystic duct is alone affected there may be no symptoms. With other malformations jaundice is a marked sign. It is present in about one-fourth of the cases at birth, and in the rest comes on in 2 or 3 days, possibly not for a fortnight. It steadily increases, and becomes very intense. The liver is enormously enlarged, and may reach the anterior superior spine of the ilium. The spleen is large. The urine becomes greenish brown, and the stools pale and offensive. Bleeding may occur subcutaneously, from mucous membranes, and from the umbilicus due to backward pressure through the ductus venosus and the left portal vein. The child suffers from flatulent distension, wasting, ascites, and œdema; and dies in about 6-8 weeks from asthenia or convulsions; occasionally life has been prolonged for 8 or 9 months. John Thomson (1892) tabulated 49 cases of *Congenital Obliteration of the Bile Ducts*, and ascribed it to a primary malformation. H. D. Rolleston (1903) added 12 more, and held that it was due to a descending cholangitis, causing primary cirrhosis and secondary obliteration; the cirrhosis being of a mixed monolobular and multilobular type. Morse (1907) stated that there are about 80 cases on record. Emanuel (1907) reported a case which supports Rolleston's view. In addition to absent ducts, rudimentary gall bladder, and biliary cirrhosis, both the spleen and the pancreas were fibrosed. Possibly the condition is due to some toxin, which is active during early foetal life, and sets up cholangitis and arrest of development. The affection may be present in many children of the same family. No doubt the name is used for many conditions, some primary and others secondary, in which absence or obliteration of the bile ducts is present at or shortly after birth. The common duct is the most frequently affected. At one end of the scale is obliteration of the common duct at the duodenal end; and at the other, complete absence of the gall bladder and all the ducts; with many intermediate varieties. Life has been prolonged for months or only for a few days. The liver is deep olive green or bronze in colour, rough, hard, and very cirrhotic in prolonged cases, and contains an excessive number of dilated bile ducts. The gall bladder is distended, small, rudimentary, or even absent. *Congenital Hepatic Cirrhosis with Obliteration of the Bile Ducts* is another name for this variety of cirrhosis. It has been found associated with retention and inspissation of bile, dilated bile capillaries, and obstruction, but no obliteration. *Cystic Dilatation of the Common Bile Duct* simulates a dilated gall bladder. It is due to obliteration of the ends of the duct, and probably of congenital origin. It is progressive from birth. Five such cases died between the ages of 13 and 23 years. In Oxley's patient the cyst held 36 oz.



**Family Acholuric Jaundice** has been described under the names of simple acholuric jaundice; congenital family cholæmia; chronic acholuric, persistent, family or congenital jaundice with splenomegaly; chronic infectious splenomegalic jaundice; and the congenital acholuric icterus of Minskowski, who reported eight cases in three generations. One of these was fatal and showed splenic hy perplasia, but no cirrhosis or angiocholitis. More than one of a family in successive generations may be affected. The symptoms are jaundice of almost indefinite duration, of a lemon yellow colour, starting in early life and possibly at birth; the presence of bile pigment in the serum and stools; and no bile in the urine. Usually there is more or less anæmia and leucopenia, splenomegaly, and to a less extent enlargement of the liver. The spleen is not invariably enlarged. The affection is compatible with long life and good health, and does not affect growth or mental capacity. Sometimes there are attacks of malaise, slight fever, diarrhœa, increased jaundice, pain over the liver and spleen, and nervous distress. Hayem found specific chorio-retinitis in some cases. It is possible that Family Jaundice may be due to simple cholæmia, splenomegalic jaundice, or biliary cirrhosis; or that these are different stages of the same affection, the result of a congenital defect in the blood-forming organs, operative through hæmolysis of red cells.

**Syphilitic Jaundice.**—The treatment of severe jaundice in the newborn, when not due to pyæmic infection, is by anti-syphilitic remedies, on the assumption that syphilitic hepatitis or peri-hepatitis may be the cause. Gubler (1882) described this variety, but few cases are on record. The jaundice is usually present at birth or comes on in a few days, perhaps not for two or three weeks. It is by no means an invariable symptom, but, when present, it becomes intense and is persistent. Occasionally it subsides and recurs after an interval. Ascites may be a prominent symptom. Hæmorrhages take place under the skin and from the mucous membranes, and the child dies in a few weeks. Recovery may take place. Congenital syphilis may be an indirect cause of *Pyæmic Jaundice* by setting up epiphysitis, which in its turn may become suppurative from secondary infection and produce pyæmia. More commonly pyæmic jaundice is due to umbilical infection and phlebitis, and inflammatory pressure on the bile ducts. It is almost invariably fatal from the pyæmia, the jaundice being a minor complication. The diagnosis is based on the fever and the symptoms of septic infection, viz., anorexia, convulsions, and coma; foul smelling pus exuding from the umbilicus, and sometimes fatal hæmorrhage when the cord drops off. Other hæmorrhages are liable to occur, and the abdomen is generally swollen and tender. The pyæmic condition is more fully described under the head of pyæmia (p. 125).



## CHAPTER XI.

### OPHTHALMIA NEONATORUM.

*Syn. : Purulent conjunctivitis—Purulent Ophthalmia.*

This disease accounts for about 10-30 per cent. of all cases of blindness—36 per cent. in the London Blind Schools (Bishop Harman); and 42 per cent. in Sheffield (Snell). Because of popular prejudices it is more common here than abroad, yet it is easily preventable.

The gonococcus is the infecting organism in from  $\frac{1}{2}$ - $\frac{2}{3}$  of the cases. Others are due to staphylococci, streptococci, bacillus coli, micrococcus luteus, pneumococcus, meningococcus, Klebs-Löffler bacillus, Koch-Week's bacillus, and the diplobacillus of Morax and Axenfeld. In many instances no organism is found.

The incubation period of the gonococcal cases has been shown, as a result of treating inveterate trachomatous pannus by inoculation with gonorrhœal matter, to be 60 hours. In the newborn it is probably less. A maternal leucorrhœa is present in two-thirds. Although the infant's eyelids are separated at the fifth month of pregnancy, they are closed during delivery, and can only become infected if the lids are separated by the fingers or instruments. At birth they are tightly closed, the skin wrinkled, and covered with fatty substance. Infection is due to vaginal secretion, sticking to the margins of the lids and the eyelashes, passing into the conjunctival sac when the eyes are open, and hence is generally due to insufficient care in washing and wiping the eyes. Congenital or ante-partum cases are due to premature rupture of the membranes or the infective organisms passing into the amniotic fluid. If the ophthalmia is not present at birth, it develops within a few hours—less than 24. Stephenson (1907) collected 19 such cases, more than half gonococcal. Commonly the eyes are infected immediately after birth; sometimes during labour; or from one to several days subsequently. A few cases may be of nasal origin.

In three-fourths of the cases it is bilateral; it is more frequent in boys, because labour is longer, and more frequent in the left eye. It begins on the second or third day, almost invariably before the fifth. Gonorrhœal infection may set up a simple catarrh. Generally it begins with slight irritation which progresses into excessive inflammatory œdema and tenseness of the eyelids; chemosis, i.e., swelling, tenseness and œdema of



the ocular conjunctiva ; and copious purulent secretion. The upper lid is red, glossy, and overhangs the lower one. The lids are gummed together and separated with difficulty. The palpebral conjunctiva is villous and scarlet, and false membrane forms in 5 per cent. A papillary form, resembling follicular hypertrophy, is not uncommon. Gradually the discharge becomes muco-purulent and finally serous, the swelling and redness subside, the granular conjunctiva becomes smooth, and the condition clears up. Even with prompt treatment the attack lasts from 2-6 weeks.

*Complications* are rare in early stages. The cornea is very susceptible in the newborn, for there are no tears to wash away the discharge. It is affected in 4-14 days. Central ulcer is more common in infants than in adults, for the epithelium is thin and becomes devitalised from stasis in the circumcorneal vessels, and then gets infected. Marginal ulcers, due to pent up discharge, are more common in later life, and may become confluent. The cornea is not primarily involved, and generally escapes, except in gonococcal infection. Ulceration begins as a circumscribed yellow spot. It increases the gravity of the prognosis, as it may lead to perforation and its sequels. Perforating abscess of the cornea may occur as late as the fifth week. Relapses are common, and the gonococcus may be found in the sac for weeks after the discharge has ceased. Arthritis of one or more joints has been recorded, and even gonorrhœal stomatitis. Arthritis may be very acute, or milder, with much effusion and pain on movement. Suppuration may occur as the result of infection by other organisms.

The *diagnosis* has an important bearing on the prognosis. In non-gonococcal cases the discharge is more often thick mucus, or muco-pus, than pure pus, and the hard tense swelling of the lids is uncommon. The gonococcus is readily stained by pyronine methylene green. Like the meningococcus and the micrococcus catarrhalis it is Gram-negative. In glucose and galactose media it forms acid. The other organisms do not ; except the meningococcus in glucose media (Mayou, 1904).

The *prognosis* is good in all cases, provided proper treatment is begun early, before the cornea is invaded. If it is clear, a favourable issue may be predicted. Under efficient treatment it should remain unaffected. There is more risk in feeble infants, because the treatment cannot be carried out as thoroughly. Permanent blindness may ensue, or patches of opaque cicatricial tissue in the cornea which may in time be replaced by clear corneal connective tissue. Two-thirds of the patients recover with unimpaired sight. Rarely cases are so serious that no treatment is successful. Streptococcal cases are rare, severe and dangerous, for the cornea may become necrotic in 12 hours.

*Prophylaxis* consists in cleanliness and the adoption of Credé's method



in all newborns. Wipe the eyes clean from maternal secretions with a damp swab, while the lids are still protected by the vernix caseosa, as soon as the head is born. Keep the water of the bath from contact with the eyes, and afterwards again wash the eyes with boiled water, normal saline, or weak Condly. If the mother suffers from a leucorrhœa, even though not known to be gonococcal, antiseptic vaginal douches should be given before delivery, and subsequently 2-3 drops of 1-2 per cent. solution of nitrate of silver should be instilled into each conjunctival sac. Credé recommended a 2 per cent. solution, but 1 per cent. is said to be efficacious, and not cause catarrh. It reduces the incidence of the disease from nearly 10 to under 0.1 per cent. (Treacher Collins). It must not be adopted for ante-partum ophthalmia, or when the inflammation has already started; nor should midwives or nurses be allowed to assume the responsibility involved in its use. The solution must be fresh, and kept in a dark bottle away from light. The great objections to its use are that it is painful, sets up a mild conjunctivitis, and medical men are loathe to adopt a system of treatment which enables parents to say that the doctor always does something to inflame the baby's eyes. It is not an absolute security, and is unnecessary in 90 per cent. of newborns. There is no stigma involved in its use, for one-third of the cases are not gonococcal. The chief substitutes are the colloidal salts of silver, protargol and argyrol, 10-20 per cent. strength, and perchloride of mercury 1 in 2000. The instillation of one drop of 1 per cent. solution of perchloride alone of all antiseptics approaches Credé's method in safety, and is quite as irritating.

*Active treatment* must be adopted at the very earliest commencement of the disease, and the reddening eye not assumed to be "only a cold." All discharge must be washed away by free irrigation with a non-irritating antiseptic solution, and a strong antiseptic and astringent applied to the inflamed conjunctiva.

The baby is placed on its back in the nurse's lap with the head towards the operator, who must sit down and hold it firmly between his knees. Separate the lids, using a retractor at the first examination, for pressure on the globe might cause rupture of a necrotic cornea. A bent hair-pin will do, if a proper retractor is not available. The cornea usually comes into view as soon as the eye is moved, if the tips of the thumbs are pressed on the extreme borders of the eye-lids and eye-ball. Dip sterilised cotton tampons in warm antiseptic fluid, and squeeze them so that the fluid drops gently into the eye without splashing, or use a douche with a sterile glass nozzle, but never a syringe for fear of injuring the cornea. Gently open and close the lids to expel retained pus. Wash until all pus is removed, and then sponge lightly with the tampon to remove coagulated masses of false membrane. Repeat the douching with plenty of fluid every 1-3 hours day and night. Use for the douche normal saline, boric acid 3 per cent., or



permanganate of potash 1 per cent. ; the last is not irritating or painful, does not injure the cornea. The discolouration can be removed by bisulphide of soda, 10-20 per cent. solution. As the case improves the strength can be reduced to 0·5 and 0·25 per cent. Other lotions are perchloride 1 in 5000, formol 1 in 2000, chlorine water, quinine 1·25 per cent., and zinc chloride 0·5 per cent., if used at the onset.

Once a day, after thorough irrigation, the lids should be everted and painted with a 2 per cent. solution of silver nitrate (grs. 10 to the ounce), avoiding the cornea. A thin film of slough is formed and soon thrown off in the discharge with many organisms. If there is very great œdema and profuse suppuration, the solid or mitigated stick of nitrate of silver may be used. It dissolves slowly, and does not reach the cul-de-sac, so a few drops of a 1 per cent. solution must also be instilled. Then wash with a salt solution to neutralise any excess of nitrate. Mild cases can be treated by the instillation twice a day of a few drops of a 1 per cent. solution ; or instil 1 drop, hourly during the day and 2 hourly at night, of a 0·25 per cent. solution, first washing away the discharge with a mild antiseptic lotion.

Another method of treatment is to irrigate the sac every half hour, and apply hot fomentations every half hour of borax (2 drachms to 1 pint) ; instilling protargol, 10 per cent. hourly, and 20 per cent. daily. Examine the cornea twice a day, and instil atropin, grs. 2 to the ounce, in addition, if it becomes hazy. Argyrol is preferred by some, collargol by others ; none of these silver compounds is as reliable as the nitrate, and they have to be used much more frequently. The amount of silver in an organic compound is no criterion of its bactericidal powers. They are useful in the early stages and in mild cases, for they are neither painful nor irritating, and can be entrusted to the nurse. Protargol is the best. It must be prepared with cold water, kept in the dark in a coloured bottle, and not used too long, as it stains the conjunctiva.

The nitrate can be painted on daily, or every other day, until suppuration has ceased, and the case passes into the stage of "ocular gleet" ; then use a lotion of zinc sulphate. The margins of the lids should be anointed with vaseline, yellow oxide of mercury, or aristol, boric acid or iodoform ointment. Ice compresses are sometimes used in early stages, but I regard them as liable to lower the nutrition of the cornea. Warm compresses are much better, and render the use of greasy ointments for the lids unnecessary.

*General Warnings.*—Strong nitrate of silver treatment must only be applied by the doctor, not in early stages, and not more than once a day. Irrigation is the most important factor in the treatment. Do not bandage up the eye. If only one is affected, the infected eye may be covered with a circular watch glass, fixed above and at the sides by plaster, but open below to allow air to enter, and 1 drop of nitrate of silver solution, 1 per



cent., instilled daily into the healthy conjunctival sac. Examine the cornea twice daily, and in everting the lids be careful not to scrape the corneal epithelium; do not use a speculum, elevators, or retractors. Instil a few drops of cocaine, 2-5 per cent. solution, before examination and treatment of the eye. Never leave pus in the conjunctival sac or in contact with the cornea. If the cornea becomes hazy instil atropin, grs. 2-5 to the ounce, t.d.s. For marginal ulcers instil eserin 1 per cent.



## CHAPTER XII.

### SKIN AFFECTIONS OF THE NEWBORN.

*Exfoliative Dermatitis—Pemphigus—Epidermolysis—Fœtal Ichthyosis—Xerodermia—Sclerema—Edema—Pigmentation.*

**Exfoliative Dermatitis** was described by Ritter in 1870. It usually begins in the second week. Following normal desquamation, which commences in a few days, and is completed in about two weeks, a superficial redness appears on the lower part of the face, and is followed by a dry scaly condition. It spreads rapidly, and the whole surface becomes of a pale red up to an intense purple colour. The skin is thickened and undergoes exfoliation in large flakes. Characteristic fissures form at the angles of the mouth. Sometimes small vesicles, due to exudation beneath the stratum corneum, precede exfoliation. The epidermis is soon regenerated, but the skin remains irritable for some time. The buccal mucosa and, occasionally, the corneal epithelium are involved. There is no fever. About half the cases die of a secondary infection, enteritis, pneumonia, or asthenia. It is either an exaggerated form of normal desquamation in the badly nourished or a severe type of pemphigus neonatorum. It may occur in epidemics, and has given rise to pemphigus by contagion (Leiner and Knöpfelmacher, 1908). It is treated by calamine lotion, zinc pastes, or boracic acid ointment; baths of bran, oak bark, or tannic acid 5 per cent. strength; and aseptic powders.

**Pemphigus Neonatorum** is an acute bullous eruption due to infection, occurring sometimes in epidemics, and conveyed by midwives. Its distribution is irregular, but the palms and soles are generally free. According to Pernet it is a bullous form of impetigo contagiosa. Both are highly contagious. Impetigo in the adult sets up pemphigus in the child, and pemphigus in the child causes impetigo in the adult. It is commonly a staphylococcal infection. Streptococci have been found in some cases, and Chuprina states that the majority are gonococcal. The infection enters through the umbilicus or the skin, and may produce acute toxæmia. The onset is generally on the fourth to the sixth day, and the mortality frequently high. It must be diagnosed from syphilitic pemphigus (q.v.).

**Congenital Pemphigus or Epidermolysis Bullosa** is a rare peculiar tendency of the skin, very early in life, and even at or before birth, to form



large bullæ as the result of trifling injury, friction, or pressure. It is often hereditary, and may be limited to one sex. The bullæ are very superficial, like those due to slight burns, scalds, and irritants; with flaccid walls and serous contents, which may be hæmorrhagic or become purulent, unless emptied when full. The parts liable to injury are most affected, especially the feet, calves, and hands. Occasionally the whole skin is involved, and becomes thin and like tissue-paper in appearance. The nails are badly formed and often lost; the tongue sometimes affected, and the teeth frequently irregular. Mental development is backward because of the physical disability; the general health is unaffected. The symptoms are limited to burning, itching, and pain. It is worst in warm moist weather, in summer; and is probably vaso-motor in origin. German-bred children seem particularly liable. It may be followed by atrophy, pigmentation, or scarring of the skin. Groups of milium-like subepidermic cysts develop round the bullæ or scars. Possibly there is a dystrophic type of case as well as one of vaso-motor origin. It continues to evolve throughout life, is comparatively benign, and resists all treatment. The health may suffer because of the mouth affection and deficient teeth. Cleanliness and protection from injury, with attention to the general health, cod-liver oil, arsenic, ergot, and tonics, are the chief measures of treatment.

**Xerodermia** is of three varieties:—(1) exaggerated dryness and roughness of the skin, with liability to squamous or fissured eczema, as in *Keratosis palmaris* or *eczema palmare*; (2) *Keratosis* or *Lichen pilaris*, a permanent exaggerated goose-flesh, in which acuminate horny projections from the follicles make the skin somewhat like a nutmeg grater. It especially affects the extensor surface of the upper arm and thigh, and is more common in girls, and in the later half of childhood; (3) *Ichthyosis*. This is a hyperkeratosis; excessive epidermal proliferation and retention of the horny layer, abeyance of exfoliation, and atrophy of the deeper layers of the corium. It is congenital, hereditary, or acquired. Byrom Bramwell reported six cases in one family of four generations, only one male, and the disease being chiefly limited to the palms and soles, parts which are usually spared or little affected. C. J. Bond (1905) found in another family that males only were affected, and females transmitted it. Oestereicher recorded the case of a woman who had three normal children by the first husband, and three ichthyotic by the second, both parents being free. The hereditary cases are usually in abeyance until some months after birth.

**Fœtal Ichthyosis** was first recorded by Richter in 1792. Lebert (1864) collected nine cases, and described a similar condition in a calf. In 1908 sixty had been recorded. It begins about the fourth month of foetal life. Most grave cases are born prematurely. The disease may be incompatible with life, or the child live for a few days or months, or it may develop at or shortly after birth. In the foetus the skin looks too small,



and is cracked and fissured. It forms irregular polygonal plates, 1-3 mm., thick, dirty white or yellow in colour, and separated by red or purplish furrows. They are largest over the back, and especially thick over the scalp and forehead, producing ectropion of the upper lid. The skin somewhat resembles irregularly cracked and fissured parchment, and forms a stiff covering, rather like the shell of a tortoise. The orifices are deepened by contraction of the skin or plugged by epidermal scales. The mouth is open, the angles fissured, and the tongue projects. The nose is represented by round holes level with the face. The scalp is seborrhœic, hair harsh and dry, and nails badly developed. The temperature is low and sweating absent. The palms and soles are unaffected; or thickened, coarse, dry, yellow, and cracked. The disease is due to increased activity of the rete Malpighii or faulty desquamation. Dumesnil and Bowen suggested that it was due to the persistence of an epitrichial layer, a membrane found by Welcker, covering the hair in embryos of lower animals, and normally disappearing about the sixth month. Against this it must be pointed out that complete exfoliation may take place after measles, or indeed every few months, and the condition re-appear. The underlying skin is moist and red, but soon becomes dry, hard, and cracked. Moreover, the acquired cases only differ in degree. The thyroid gland has been found atrophied, absent, or normal. The severity of the affection, the development of the child, and the duration of life are variable. The development of the nose, ears, fingers, and toes is interfered with mechanically. Many remain small and delicate, some die early from a kind of toxæmia, a few show infantilism, while others develop normally though the disease is well marked. As a rule the prognosis varies directly as the extent of the disease. They should be treated by persistent massage with oil and weak mercurial ointment. Systematic exfoliation must be encouraged by resorcin dr.  $\frac{1}{2}$ -1, glycerin or almond oil oz.  $\frac{1}{2}$ , and glycerin of starch or lanolin oz.  $1\frac{1}{2}$ , applied once or twice a day, and daily washing with a superfatted soap. Thyroid extract and pilocarpin are useless.

*Ichthyosis Herpetiformis* (Bielt's Bands) is a variety of Ichthyosis in which the patches are in streaks and the unaffected parts of the skin remain healthy. It is limited to one child in a family, rare on the scalp, uncommon on the neck and face, of no special nerve distribution, and usually unilateral. These congenital streaks or bands of thick rugose skin may resemble moles, or, more often, patches of rough unwashed skin, for the dry papillary outgrowths become almost black with dirt. They can be destroyed by cautery when the child has grown up.

*General Treatment of Ichthyosis:* Polish down rough and scaly projections, and stimulate the secretion of sweat and sebum. Give a warm alkaline bath, dry, and rub in diluted glycerine of starch, salicylic acid ointment, gr. 5-10 ad oz. 1. For bad cases use a stronger ointment of salicylic acid and resorcin. Internally try thyroid extract and cod-liver oil.



**Sclerema Neonatorum.**—This rare affection, known in the time of Galen, was first described by Uzemberius (1718). It is more common in France and Italy than elsewhere. It is also described under the names of Sclerema Œdematosum and Sclerœdema; and it must be distinguished from Sclerema Adiposum or Symptomatic Sclerema, also sometimes called Sclerœdema or Œdema Neonatorum. It occurs at birth or shortly after, not later than six months. It is most frequent in illegitimate children, foundlings, the weak and premature, and in those of women who have suffered privation during pregnancy. It is not seen in children of the better classes, is not due to syphilis, and apparently is a result of intra-uterine malnutrition, or the solidification of subcutaneous fat on account of feebleness and rapid loss of heat. No less than three cases have been reported in twins during the last few years. A mild type is not very rare in Great Britain. It is confined to the skin and subjacent tissues, may affect normal full-time infants, and ends in recovery at about 6 months. The severe variety is more prevalent on the Continent, in the weakly and the premature, and produces serious constitutional symptoms, especially in the alimentary, respiratory and renal organs. It is characterised by an induration, a progressive thickening and hardening of the skin and subcutaneous tissues, which may or may not pit on pressure according to the degree of infiltration. The parts are doughy or hard, and may be painful on pressure or handling. There are no definite histological changes. The induration is progressive, but does not involve the joints. It may be limited or general, in patches which coalesce, and cause general rigidity. It especially affects the gluteal regions, legs and calves, the deltoid and pectoral regions, the back and back of the neck, the cheeks, and may spread on to the abdomen. The chest is affected late or not at all; the face may escape, thus enabling the babe to continue suckling; the penis, scrotum, palms and soles are unaffected. The child is generally badly nourished, weak and somnolent, with a subnormal temperature, even down to 85° F., superficial and weak breathing, and almost inaudible cry; and dies from asthenia or coma, occasionally from broncho-pneumonia or convulsions. The skin feels cold and is adherent, and cannot be raised from the subjacent tissues, nor these from those below. It is usually board-like, smooth, and regular, but may be roughly nodular, with a waxen, yellowish, red, or bluish discolouration. On pressure the colour fades to white, and slowly returns, and there is rarely pitting. The condition has been aptly compared to that of a half frozen corpse or a flitch of bacon. The limbs become rigid and motionless, and the face mask-like. Respiration and circulation are feeble, and the lips blue. There is no albuminuria. Obstinate constipation has been ascribed to serous infiltration of the intestinal mucosa. The prognosis depends upon the extent, the general condition of the child, and the temperature. It is worst if the temperature is low and the thorax or face involved. Atelectasis is often



present. Many die in 3 or 4 days, but a few have recovered (70 per cent. Clementowsky). In mild cases extensions may take place, and clear up irregularly and continuously. The treatment consists in the use of an incubator, systematic hot baths, and massage with olive oil or a weak mercurial ointment.

**Œdema Neonatorum** presents the ordinary signs of œdema, pits on pressure, and affects the penis and scrotum. It is due to debility, cold, and venous congestion. The most common sites are the dorsum of the feet and hands, and the eyelids.

*Sclerema Adiposum* or *Symptomatic Sclerema* is characterised by a stiffness of the skin. It is due to loss of fluid from the body in prolonged illness, acute diarrhœa, and some forms of pneumonia.

**Congenital Pigmentation of the Skin.**—For over 150 years Japanese physicians have noted that the infants of their race present, at or soon after birth, one or more bluish pigment patches, darker in the centre, in the sacro-gluteal region and over the back. They vary in size, and may be found on the extensor surfaces of the extremities. These patches disappear in a few years, and rarely persist up to puberty or throughout life. They are found in negroes, and in many Asiatic races of mixed descent, and have been regarded as evidence of negroid ancestry. Similar spots were demonstrated in a white baby by A. Edmunds (1906), and have been discovered in pure German infants. Probably they represent an earlier stage of evolution, or an atavistic tendency. They are more marked in males than females.



## CHAPTER XIII.

### DISORDERS OF METABOLISM.

*Acholia—Lithæmia—Acidosis—Delayed Anæsthetic Poisoning—  
Recurrent Vomiting—Diabetes—Scurvy.*

**Acholia.**—Under the name of “*Cæliac Disease*,” or *Belly Affection*, Gee (1888) described a condition in infants in which the stools contain little or no bile. Cheadle (1903) named it “*Acholia*.” According to the degree of deficiency in bile, the stools vary from pale straw colour, clay colour, dirty grey like putty or porridge, to the whiteness of white paint or pipe-clay. They are much the same as those seen in obstructive jaundice, but there is no yellowness of the skin or bilinuria. They are acid or slightly alkaline; glistening, greasy and fatty; often frothy; very offensive and larger than normal; and slightly loose, or like a mass of dirty grey or white paint.

The size of the stools is due to defective absorption; the offensive odour to the decomposition of excessive fat; and the absence of bile to deficient secretion of bile, or to lack of conversion of bilirubin into the hydrobilirubin of the fæces, from occlusion of the pancreatic duct. Most probably it is due to deficiency of bile, for in the most marked cases there are no bile acids in the stools. Both a decrease and an increase of the urea and leucin have been found in the urine. No definite changes are seen after death. The liver is normal in appearance and the spleen may be enlarged.

These cases occur under 5 years of age, usually under 2. Sometimes the attacks are preceded by coldness of the extremities, and can be warded off by keeping them warm. There may be a little fever at the onset. The child is irritable, fretful, languid, flabby and pale. The appetite fails, vomiting is not uncommon, and the stools are as above described. The abdomen is full, flaccid, and distended by gas. Progressive wasting and a subnormal temperature are constantly present. The course is chronic. It is liable to relapse but rarely proves fatal.

The causation is doubtful. From the fact that it is apt to arise during the period of dentition, it has been ascribed to difficult and painful teething, which is supposed in some way to cause reflex irritation of the liver. More probably it is due to chill and overfeeding.



The chief difficulty in diagnosis is its similarity to abdominal tuberculosis. Some undernourished infants, overfed on cow's milk, pass almost white stools. These are not acholic, but are due to a perversion of liver function. Bilirubin is converted into a colourless urobilinogen instead of into urobilin. Urobilinogen is derived from hydrobilirubin and forms a colourless compound in an alkaline solution. Possibly this is the true explanation of the colour of the stools in the cases described as acholia.

In treating these cases it is essential to reduce the work of the liver and digestion. Fats and starches must be given in small quantities only, and predigested. Milk may have to be stopped. Malted foods are useful. For looseness of the bowels give bismuth and soda; salol and sulphocarbates for offensive smell; chloral and bromide to allay nervous irritation. As hepatic stimulants give brandy, tincture of podophyllin, sodium salicylate, and ammonium chloride. Glycocholate of sodium may be tried as bile salts stimulate bile secretion. Small doses of grey powder are also useful; and iron and arsenic during convalescence. Cod-liver oil is generally injurious, and the seaside rarely beneficial.

**Lithæmia.**—Lithæmia is a common functional disorder which probably depends on disturbance of the normal activities of the liver. No one name is absolutely appropriate, for the symptoms are due to different kinds of toxic products, resulting from the defective metabolism of protein in several organs and the non-excretion of various retrograde products. It is called *Lithuria*, because of the excessive elimination of urates in the form of pink amorphous urates, or of uric acid crystals as a brick-dust deposit. The mere precipitation of uric acid crystals must not by itself be regarded as proof of excess of uric acid, for it is often due to mere concentration of the urine. This is indicated by high specific gravity. Lithuria is not a specific disease, nor limited to disturbance of liver functions. It arises from a variety of conditions associated with disturbance of nutrition. Temporary attacks are common, and may be due to excessive production rather than abnormal elimination.

Lithæmia is common in children, especially those of a gouty or neurotic constitution. The attacks come on suddenly after errors in diet, excess of nitrogenous food, or exposure to cold winds. Many can be traced to injudicious feeding on rich dishes and sweets; or to over-stimulation of the nervous tissue by brain fatigue, amusements and late hours. Regular periodical attacks occur in some children, and perhaps explain sick headache and migraine.

The child is fretful, irritable, and complains of headache and languor. The complexion is pale and sallow, with a slightly yellowish tinge and dark rings under the eyes. The tongue is furred and the breath offensive; the appetite variable, capricious, and perhaps excessive; the bowels confined, and the stools deficient in bile.



The treatment consists in an immediate dose of calomel or grey powder, followed by a saline purge if needful, and limitation of the diet to plain easily digested food, allowing no excess of sweets, meat or extractives. Tea and coffee must be forbidden, and alkaline or aerated waters given freely. Occasional small doses of calomel should be given for 2-4 weeks; and a mixture of tincture of nux vomica with bicarbonate of soda or with dilute nitrohydrochloric acid, to assist digestion.

**Acidosis.**—*Syn.*: *Acidæmia*—*Acetonæmia*—*Aciduria*—*Acetonuria*—*Acid Intoxication*.—Acidosis may be defined as an abnormal metabolism of carbon leading to the appearance of organic acids in the blood and urine, and the formation of ammonia to neutralize these acids. The normal chemical reaction of the tissue fluids is faintly alkaline, because the basic ions slightly exceed the acid ones. In acidosis the tissue fluids become less alkaline. The organic acids, or acetone bodies, are diacetic and B-oxybutyric acids, and are derived from fat imperfectly metabolised in the liver and tissues. They are rapidly neutralized by sodium in the tissue fluids, potassium in the cells, and by alkaline earths chiefly derived from bones. If this is not enough, the excess is neutralized by ammonia, derived from protein, which would normally have been metabolised into urea. Should more acid be produced than can be thus neutralized, the reaction of the tissue fluids may be altered and cause symptoms of acid intoxication. It is, therefore, evident that acidosis may be very common, though acid intoxication is rare. It can exist long before the tissue fluids are rendered less alkaline, and before toxic symptoms arise. One passes gradually into the other. But acetone bodies are not poisonous, so some further factor must be sought for to account for acid intoxication.

*The diagnosis of acidosis.*—An excess of organic acids, not normally present in the urine, is excreted. If more are produced than can be neutralized, the urine is hyperacid. The breath and urine have the peculiar smell of acetone, and acetone bodies are found in the urine. Acetone, diacetic acid, and B-oxybutyric acid appear consecutively in the urine, in accordance with the increasing failure in the oxidation powers of the tissues. When acetone is heated to 100° C., it is split up into diacetic acid and carbonic acid. Hence, no satisfactory means have been devised for estimating the two bodies separately in the urine. Small amounts of acetone in the urine are unimportant, and may be found in the healthy. Large amounts and the presence of the other bodies are pathological.

*Tests for Acetonuria.*—Add 10 drops of caustic soda solution to the urine, and 20 drops of fresh solution of sodium nitroprusside. A bright red colour is produced. If this is due to creatinin it is discharged by adding glacial acetic acid. If acetone is present the colour turns dark claret or purplish. In the absence of creatinin, acetone causes a carmine-like colour, becoming purplish on adding acetic acid. With ammonia, instead of caustic soda, the colour varies from magenta to deep violet. If the urine is boiled first the acetone and diacetic acid are volatilised and driven off, so the colour reactions fail.



The usual test for diacetic acid is the addition of solution of perchloride of iron, which changes the colour of the urine to red or reddish purple. Salicyluric acid causes a similar colour reaction, but it is not prevented by boiling the urine.

If 2 drachms of bicarbonate of soda are taken by a normal individual the urine becomes alkaline for 24 hours or so. In acidosis, the sodium is seized on by the acid radicles and passed out as neutral salts, and the urine remains acid.

Normally, too, the ammonia-N in the urine is inconsiderable, but in acidosis the ammonia excretion is much increased.

As a final test, the reduction in the alkalinity of the blood must be mentioned.

Acetonuria is undoubted evidence of acidosis. It does not occur until the acidosis is of such a degree that the acid products of metabolism are no longer fully neutralized before excretion. The absence of acetonuria does not disprove the presence of acidosis, but it is unlikely that serious symptoms of acid intoxication can occur without such evidence. Another point of some importance is the occurrence of variations in the total acidosis without a corresponding change in the colour reactions in the urine.

*The Conditions under which Acidosis occurs.*—It is more common and more easily produced in children than in adults, and in women than in men. Acetone may appear in the urine in the apparently healthy. It is regarded as a constant and unimportant constituent in children by Baginsky. Metabolism is less stable, and the reserve of carbohydrate in the liver is smaller.

It is found in starvation and cachexia; on the omission of carbohydrates from the diet; as the result of the excessive ingestion of fat; in pyrexial states, such as scarlatina, pneumonia and broncho-pneumonia; in severe vomiting and diarrhoea, gastro-intestinal disorders, epidemic diarrhoea, appendicitis, acute peritonitis, and intestinal obstruction; in eclampsia, tuberculous meningitis, and nervous disturbance due to fright; in poisoning by morphine, salicylates, phosphorus, and phloridzin; after anæsthesia; in hepatic inadequacy, acute yellow atrophy, recurrent vomiting, and diabetes. Acetonuria must be regarded as a sign of faulty metabolism. It may be acute or chronic.

In considering the above causes it is obvious that one or more factors in the production of acidosis may be present in the particular case. The deficient oxidation in pneumonia, the auto-intoxication of intestinal disorders, the toxæmia of scarlatina, sepsis and appendicitis, and the delayed oxidation and fatty changes the result of poisons may all be invoked as explanations of the acidosis. On the whole our present knowledge indicates that it is due to:—

- (1) An excess of fat in the diet, or a superfatted liver with hepatic inadequacy in the metabolism of fat and carbohydrates. A fatty liver may be due to excess of carbohydrate food, or to infiltration with unsaturated fats which are fully oxidised in the muscles and tissues.
- (2) A deficiency of, or inability to make use of, carbohydrates in the diet.



- (3) The effect of toxins or other poisons on the metabolism of the liver, which may or may not be overloaded with fat. This is the most general explanation.

Acetonuria may occur without symptoms. Acid intoxication is commonly a complication of other diseases, and occasionally occurs independently, as in delayed anæsthetic poisoning and recurrent vomiting.

*The Pathogenesis of Acidosis.*—Acetonuria is increased by the addition of lower fatty acids, and fat to a less extent, to the diet. It is decreased by adding protein and abolished by adding carbohydrates, for they act as fat-sparers, reduce the amount of fat used, and aid its complete metabolism. Acetone is due to the incomplete combustion of fat into carbonic acid and water. Diacetic acid is a combination of acetone and carbonic acid, and on deoxygenation forms B-oxybutyric acid. Acetone can be formed from gelatin, leucin, and albumin *in vitro*. Possibly it can be formed in the body to a limited extent from protein, but the amount cannot be much for the nitrogen and sulphur are not increased in the urine to the extent which would be the case if this were the source.

Carbon leaves the body as carbonic acid, neutral salts, or combined with nitrogen in the urine. Abnormally it may appear in the urine uncombined with nitrogen, as sugar; that is, unkatabolised. Or it is abnormally katabolised, and appears as acetone.

The complete combustion of fat appears to be dependent on an adequate simultaneous consumption of carbohydrate to supply the necessary oxygen. A deficiency of carbohydrate alone may be sufficient to cause defective oxidation and metabolism of fat, and acidosis. This may account for the acidosis in all states of starvation; and in diabetes, in which must be considered in addition the effects of an excess of fat in the diet, possibly some deficiency in the oxidising power of the body cells, and perhaps some breaking down of protein as well.

Alkaline soaps are formed in the intestines from fat in the food. If an excess of fat is ingested, alkalies, which would otherwise reach the tissues, are used up in the intestine, and are consequently not available for the neutralization of the inorganic acid end-products of metabolism. Under such circumstances the alkalies in the cells, tissue fluids and bones are utilised as far as possible. Finally, ammonia is formed to neutralize these end-products, and appears in excess in the urine. Thus, an excess of fat in the diet does not lead to an abnormal production of organic acids in the body, but does cause an excessive loss of alkaline bases by the intestinal tract in the form of soaps, sometimes in such excess as to reduce the alkali balance of the body.

The normal ammonia excretion in the urine is about 5 per cent., but in sick children it may reach 30-50 per cent. of the total N-excretion. It can be increased by adding fat to the diet. Such an increase can be reduced to *nil* by the reduction of fat ingested, and the administration of



sodium bicarbonate, and other alkaline bases. Folin found it increased on a diet of starch and cream, an excess of inorganic acids being ingested. The ammonia excretion is also increased if the patient has to live on the protein and fat in the tissues, as in severe vomiting and diarrhœa, the toxæmic vomiting of pregnancy, starvation of various kinds, and lack of carbohydrate food. The increased excretion is due to the fact that the body has to manufacture ammonia to neutralize the acid end-products of metabolism, which are normally neutralized by alkaline bases. If there is sufficient alkali available the ammonia is converted into urea. Most of these acid end-products are organic acids. Some are formed by the oxidation of phosphorus or sulphur from broken down protein.

Excess of fat in the diet causes *Fat Diarrhœa*, a relative increase in the amount of neutral fat in the fæces. It gives rise to increased soap formation, and the withdrawal of alkalies and earthy salts. Lime salts and alkalies are also found in excess in the fæces during fever. In acid dyspepsia an excess of free fatty acid is found in the fæces. And, finally, it increases the ammonia in the urine, an increase which disappears on the exclusion of fat from the diet.

We are justified in concluding that a relative acidosis may be simply the result of an excess of fat in the diet, in consequence of an excessive loss of alkaline bases by the alimentary canal, without an abnormal production of organic acids in the body. The extent of the ammonia excretion is a measure of the degree of acidosis. A high ammonia excretion may be due to an insufficient intake of fixed bases, and is then cured by giving bicarbonate of soda.

*The Liver and Acidosis.*—The appearance of ammonia in the urine is not due to any diminution of the urea-forming function of the liver. Even very sick children can convert ammonia salts into urea. In some cases degeneration of the liver is present. Richardson and Howland have brought forward evidence that in recurrent vomiting there is a deficiency in the oxidising power of the liver and body cells. In post-anæsthetic cases, and those of recurrent vomiting, the most striking anatomical feature is the superfatted liver. The heart and kidneys are also fatty. A liver may be intensely fatty without evidence of disturbed function; but acidosis and acid intoxication may occur. The metabolism of fat is an important function of the liver.

A superfatted liver results from various causes:—

- (1) An actual excess of fatty food; or a relative excess and defect in the capacity to metabolise fat.
- (2) An excess of carbohydrates; or a relative excess and defect in the capacity to metabolise carbohydrates, e.g., recurrent vomiting.
- (3) Deprivation of carbohydrates, as in starvation. The fat is carried from the other tissues to the liver in order to be elaborated for oxidation and utilization in the system generally.



(4) Sepsis, specific organisms and toxins, poisons and anæsthetics.

These lead to further overloading of the liver and check oxidation.

The storage of fat may be absolute or relative to the metabolic powers of the tissues, or due to the mobilisation of fat in the liver as the result of starvation or auto-intoxication. It probably precedes acidosis and acid intoxication. The characteristic liver is found in post-anæsthetic cases and in recurrent vomiting. Possibly it is the effect, and not the cause, for a great accumulation of fat in the liver can appear and disappear with remarkable rapidity, in a few days or hours, certainly within 24 hours. But it is difficult to believe that such a profound change could be produced by half a drachm of chloroform, as seen in some anæsthetic cases.

Probably the fatty liver is only dangerous as a predisposing cause, in that it implies defective metabolism and oxidation. Then a further perversion causes imperfect oxidation and toxæmia due to the breaking down of all the hepatic functions, proteolytic, glycogenic, and antitoxic. On the other hand, a very small portion of normal liver seems to be sufficient to carry on its functions. In the post-anæsthetic cases the drug must be regarded as the last straw. It has been shown experimentally that chloroform, and ether to a less extent, increase the fat in the liver of dogs, and that the heart and kidneys are also affected through autolysis.

Acetone bodies are found in the urine and stomach for some hours after prolonged anæsthesia. The severe vomiting is probably due to acidosis, and may be relieved by bicarbonate of soda by mouth or lavage of the stomach therewith. Such vomiting exaggerates the symptoms, for severe vomiting aggravates acidosis, causes serious loss of fluid, and disposes to acute degeneration of the liver. Thus, acute yellow atrophy and severe vomiting, with increased ammonia excretion, may occur in pregnancy. In anæsthesia oxidation is checked by the preliminary starvation, enforced rest, mental disturbance, narcosis, and the anæsthetic. It may, therefore, be argued that the post-anæsthetic cases are true cases of anæsthetic poisoning, or that they are due to acid intoxication in a predisposed child, the anæsthetic acting as the final straw.

Thus, there are two definitely associated factors, viz., a morbid state of the liver, a fatty infiltration with sometimes areas of necrosis, and manifestations of acid intoxication. Both the fatty liver and acidosis may occur independently of toxic symptoms, and the state of the liver may precede or be secondary to the acidosis.

*Acid Intoxication.*—Acetone bodies are not poisonous, so we must seek for some additional factor as the cause of the symptoms of poisoning. Probably fatty acid intoxication alone cannot cause death.

It is not due to an insufficient supply of alkaline bases to neutralize the acids formed, for the alkalinity of the blood may be even lower than in diabetic coma without any of the symptoms of intoxication (Benedikt). It must not be ascribed to personal idiosyncrasy, for in anæsthetic cases



a similar anæsthetic may have been taken previously without ill effect. There is no evidence of poisoning by carbonic acid. Diabetic coma can be produced by the abrupt omission of carbohydrates from the diet and stayed by administering them, but the clinical evidence in post-anæsthetic cases does not support the theory of carbohydrate starvation.

Eustace Smith has described as "food fever" cases of recurrent pyrexia, vomiting and gastro-intestinal disturbance. He regards chill as an exciting cause, and ascribes them to an excess of carbohydrate food, fermentation, gastro-intestinal catarrh, and intestinal auto-intoxication. In some respects they are analogous to cyclical vomiting. Many children with this latter affection crave for and eat much carbohydrate food; possibly because of their inability to make use of it. An excessive fattening diet, cod-liver oil and such like, produces an intensely fatty liver, something like *foie gras*. The defect may be a hepatic inadequacy to deal with more than a small amount of carbohydrate or fat. Some cases are cured by a simple mixed diet.

In the anæsthetic cases it is possible that the culminating effect is due to the destruction of oxidising enzymes in the liver. Possibly the additional factor is a toxæmia of intestinal origin or due to the autolysis of liver cells. In cyclical vomiting Howland and Richards found excessive indicanuria, increased excretion of uric acid, especially at the onset; decrease in the excretion of sulphuric acid, and increase in that of unoxidised sulphur; and acidosis. The excess of uric acid could not be due to excess of exogenous purins, and must consequently be due to deficient oxidation of endogenous ones, which are normally oxidised to the extent of 50 per cent. in the body. It, therefore, showed increased tissue destruction. The excess of unoxidised sulphur is further evidence of deficient oxidation. Lactic acid was also found in the one instance in which it was sought for. It is an intermediate product in the combustion of dextrose, and its presence was additional evidence of deficient oxidation.

The excess of indican showed increased intestinal putrefaction. Indol is oxidised into indoxyl, and this combines with sulphuric acid to form indoxyl-sulphate or indican. Skatol is a methyl-indol formed in an analogous manner. Phenol combines directly with the sulphuric acid. By experiments on animals these observers obtained some interesting results. Decreasing the ability of the cells to utilise oxygen, by inhalation of chloroform or injection of potassium cyanide, and giving measured doses of indol and phenol, they found that the non-toxic indol and phenol became toxic, or if toxic were much increased in virulence. Marked gastro-intestinal congestion and necrosis, and degeneration of the liver were produced in dogs. Hence under conditions of imperfect oxidation, indol, and probably skatol and cresols, become toxic. Constipation is a factor in so far as it increases absorption. It must be mentioned that hydrocyanic acid interferes with ferment action, and might be the cause of some of the results.



*Symptoms.*—Although involving a certain amount of repetition, for the symptoms ascribed to acid intoxication occur in several affections described subsequently, it is advisable to give a concise summary. Vomiting is incessant, sometimes hæmorrhagic or coffee-ground in character. It may occur without nausea, or there may be nausea and excessive retching. Intense thirst, constipation or diarrhœa, and some degree of epigastric pain and tenderness are present. Sometimes there is slight jaundice, usually towards the end. Air-hunger, restlessness and sleeplessness are noticeable. Irritability alternates with dulness and apathy. Prostration is great and emaciation rapid. Delirium and convulsions, or drowsiness and coma end the scene. Or attacks of collapse, perhaps fatal, ensue.

The eyes are bright and sunken, the face pinched and drawn, and pale or flushed in early stages. The breath smells of acetone. The tongue is often clean at first, and later becomes dry and furred; the mouth being dry and covered with viscid mucus. The skin is dry and inelastic. Occasionally measliform, urticarial, or purpuric rashes develop. The abdomen, and sometimes the head, is retracted. The pulse is rapid, weak, and apt to be intermittent or irregular. The temperature may be normal, but more usually is raised, seldom above 102° F. Occasionally there is hyperpyrexia. Acetonuria is not invariable, for the acetone bodies may not be eliminated by the kidneys in some of the anæsthetic cases and early stages of recurrent vomiting. In diabetes they may decrease before the onset of coma. Urea excretion is variable, usually decreased, while the ammonia-N is increased. It is noteworthy that the symptoms of post-anæsthetic cases resemble those of acute yellow atrophy, and that acute yellow atrophy is an occasional cause of death after operation. Generally the liver shows extensive fatty infiltration.

*Treatment.*—Encourage excretion by diuretics, diaphoretics and purgatives, if they can be retained. Wash out the stomach and put in sugar solution. Saline injections are useful. Give fluids freely. Omit fat from the diet. Try rectal feeding. Caffein, ammonia and strychnia are the best stimulants. Alkalies are given freely, up to 180 grs. of bicarbonate of soda daily, in order to relieve the strain on the ammonia compensation for acidosis. If they are not retained by mouth give them with salt by rectum. Experience has shown that they are not of much value, unless the supply of base to neutralize the acids has failed. If the cause is toxic, an autolysis of liver cells leading to defective protein metabolism, alkalies are unlikely to do good. The cells can utilise carbohydrates well, but oxidise protein badly and fat even less. On the assumption that the carbohydrates have been used up and a state of starvation is produced, it is worth while to give dextrose freely.

**Delayed Anæsthetic Poisoning.**—*Syn. : Delayed Chloroform Poisoning—Fatty Liver and Death after Anæsthesia.*—It is to Guthrie that the credit is due of establishing the connection between anæsthetics, fatty infiltration of the liver, acidosis, and the severe



or fatal illness which sometimes ensues on the administration of anæsthetics to apparently healthy children. His first paper was published in the *Lancet* in 1894. As long ago as 1850 Caspar suggested it as a cause of death, and Langenbeck found a fatty liver in a fatal case. Three cases were reported by Bastianelli, and one by Thiem and Fischer, in 1890. Fraenkel investigated five in 1892. Now there are quite a hundred on record.

Most of the cases have followed the administration of chloroform. A few have been due to ether, ethyl chloride, and nitrous oxide. The halogen group is present in both chloroform and ethyl chloride. The amount of the anæsthetic and the duration of the operation seem to have little bearing on the production of the affection or the degree of acetonuria. Patients have developed it after less than two drachms of chloroform in an operation lasting seven minutes, and after ethyl chloride and the removal of adenoids in about a minute.

The patient may seem to have quite recovered from the anæsthetic. Then in about 12 hours, or at any rate within 36 hours, the child becomes restless, tosses about, struggles, grinds its teeth and utters piercing shrieks. It is pale or flushed, looks anxious or terrified and has bright red lips, which may become cyanotic later. The pupils are often dilated, sometimes unequal, sometimes small. The pulse is frequent and breathing accelerated. The breath smells of acetone. Vomiting is almost invariable, frequent, copious and continuous. The vomitus sooner or later resembles coffee grounds or the dregs of beef tea, or may be quite black. Thirst is sometimes intense.

Or the onset may be characterised by drowsiness and apathy. In some instances vomiting and restlessness are the only symptoms throughout. The cerebral symptoms may take the form of maniacal delirium, much like that of acute yellow atrophy of the liver. They may be suggestive of acute meningitis or diabetic coma. Apathy and delirium may alternate. Consciousness is generally lost early and remains absent. Occasionally it returns in intervals of apathy and dulness.

Incontinence or retention and partial anuria occur, with acetonuria, albumin and casts. The chance of recovery is less in the absence of acetonuria. Headache and pain are absent; or there may be slight epigastric tenderness due to the vomiting. The temperature varies from normal to hyperpyrexia, and is usually irregular. Air-hunger and cyanosis are frequent. Jaundice and hæmorrhage into the lungs have been reported.

The pulse becomes frequent, running and uncountable. Vomiting becomes less frequent and often ceases. Unconsciousness passes into coma. The circulation and breathing gradually fail. Death ensues from asthenia, and sudden or gradual cardiac or respiratory failure, perhaps during an attack of black vomiting. Consciousness may persist until the end.



Death commonly takes place in 24-48 hours after operation ; in one case not until after 8 days. Even severe attacks, with much hæmorrhagic vomiting, may be recovered from.

*Morbid Anatomy.*—The liver is generally, but not necessarily, enlarged. Its colour is a pale uniform fawn, buff or canary yellow, studded with purplish spots due to the intralobular veins. The capsule is not thickened. Its substance is firm and brittle, and much oil can be scraped from the surface on section. The whole organ is affected, and shows, microscopically, an extensive fatty degeneration and infiltration. The colour is due to the excess of fat and not to bile staining of necrotic liver tissue, such as is seen in acute yellow atrophy and after phosphorus poisoning, in which the amount of fat is diminished. Cloudy or fatty degeneration is also found in the kidneys, heart, muscles and gastric mucosa. The fatty degeneration may be more marked in the kidneys than the liver. Most of these changes have been found after chloroform anæsthesia, but they may be due to other anæsthetics.

*Pathogenesis.*—It has been stated that acidosis is common in children ; that it is frequently present after anæsthesia and may account for the after-vomiting ; and that it is often associated with a fatty liver. A similar fatty change can be produced experimentally in animals by chloroform, and to a less extent by ether. Even admitting that a fatty liver can be thus produced, the fatal cases, after the short period of anæsthesia induced by ethyl chloride and nitrous oxide gas, suggest that the fatty change is not due to the drug. Sepsis is a cause of extremely fatty liver, and many of the cases have occurred after operations for septic conditions.

Probably there is a pre-existent fatty liver, functionally inadequate or on the verge of inadequacy, and the action of the anæsthetic, combined with the effects of preparation for the operation and the shock thereof, proves the last straw. The excretory power of the liver and kidneys is decreased by the anæsthetic ; more by chloroform than by ether. Though there may be acid intoxication, it is probable that the fatal factor is a toxæmia dependent on disturbance of the proteolytic and antitoxic functions of the liver, for the symptoms are more suggestive of acute yellow atrophy than of diabetic coma.

The excretion of acetone depends on idiosyncrasy, its previous existence in the urine, and the nature of the anæsthetic. Sepsis, constipation and nephritis delay excretion. In chronic acetonuria the excretory organs are trained in its elimination. Acute acetonuria, such as is developed in acute infective conditions, e.g., appendicitis, is much more dangerous. Chloroform hinders its excretion more than ether. Both drugs induce an acute temporary acetonuria, which may be fatal if the excretion is deficient. Yet, up to the present, it is not certain that the acid intoxication is the cause of the symptoms. The signs thereof in the urine may have to be regarded merely as indicative of inadequacy of the liver, and that autolysis



of the liver is actively proceeding. On the other hand, it is not absolutely proved that the fatty changes in the liver are the primary cause. Such changes are more common in chronic suppurations and tuberculosis than in acute suppurations, and one would expect delayed anæsthetic poisoning to be more common in these cases if the fatty liver is to blame. Experience does not confirm this expectation. It is quite possible that the fatty change can be accounted for by the action of an anæsthetic, even in small doses though sufficient to produce anæsthesia, on tissues predisposed to acute fatty degeneration by some form of toxæmia.

In the diagnosis it is important to exclude fat embolism, acute yellow atrophy, acute septic intoxication and poisoning by antiseptics, notably carbolic acid and iodoform, causes to which the affection has been ascribed in the past.

*Treatment* is essentially prophylactic. Highly neurotic and fat children are most liable. So, too, those who are subject to bilious attacks or acute gastric catarrh. Examine the child for fatty liver and acetonuria, and do not give chloroform if either of these conditions is present. No anæsthetic should be permitted during acute acetonuria; ether should be used by preference in acute infective states. In preparing a child for operation reduce or omit the fat in the diet, and protect from fright and mental anxiety.

Once the symptoms have arisen the treatment is that of acid intoxication.

**Recurrent Vomiting.**—*Syn. : Fitful, Cyclical, or Periodic Vomiting—Lithæmic or Bilious Vomiting—Vomiting with Acetonæmia (Marfan)—Migrainous Gastric Neurosis (Rachford).*—Occasionally cases of recurrent vomiting are seen. They are not due to mechanical causes, nor associated with dietetic errors, nor with abdominal pain beyond what is due to the strain of the vomiting. Often these children are fed with an excess of caution.

Lombard described the affection in 1861. Gee reported some cases in 1882 under the title of “Fitful and Recurrent Vomiting.” Leyden in the same year described it as periodic vomiting.

*Etiology.*—It is most common among nervous, highly-strung, precocious children of the upper classes, with a hereditary history of neuroses, gout or migraine. Occasionally it affects several children in the family. It is rather more common in girls than boys, and in the winter months. The most common age is the sixth to the eighth year, but it may have begun much earlier. Rachford states that in one instance it began in the third month of life. Certainly it may occur in the first year, and persist up to puberty. As a rule it does not begin before the third year or after the sixth. Indoor life, constipation, excitement, overwork and fatigue are both predisposing and exciting causes. Overeating and fright are excitants.



*Symptoms.*—There may be a prodromal period characterised by anorexia, offensive breath, furred tongue, nausea, gastric discomfort and constipation; dark rings under the eyes and sallow complexion; malaise and headache; lassitude and sleepiness, or nervous irritability with twitching of the eyelids, stammering, restlessness, and sleeplessness; dyspnœa and sighing respirations, and fever. Some attacks are preceded by the passage of almost white stools or by watery diarrhœa. A few have been ushered in by convulsions. More commonly in the midst of perfect health, even while in bed and asleep, without any definite exciting cause, without any error in diet or disturbance of intestinal action, the child is suddenly seized with vomiting. Nothing is kept down, not even water. Food, acid, mucus, bile and possibly blood (bright red or coffee-ground material) are brought up. Snow reported an excess of free hydrochloric acid and mucus, apparently pure gastric juice, in four cases. The vomiting is frequent, perhaps every quarter of an hour for twelve hours, projectile, without any special nausea, and does not become fæcal. It may be accompanied or replaced by severe retching. Perhaps there is a burning pain in the epigastrium, or a “nasty” substernal pain.

The breath smells of acetone, or may be offensive. The tongue is clean, or more or less coated. Thirst is sometimes present and intense, the lips being dry, and the tongue parched and cracked. The eyes rapidly become sunken, expression anxious, and the prostration great. Wasting is rapid and extreme; the abdomen cariniform, retracted, and markedly hollowed. A girl of 11 years weighed only 24½ lbs. (Shaw and Tribe). Undue pigmentation has been noted. The appetite may be retained, or there may be complete anorexia. Sometimes there is a little epigastric pain and tenderness. Constipation may resist all treatment and, when the bowels are moved, the stools are very offensive. The breathing is of the air-hunger type, sighing, or rapid and panting. The pulse is generally rapid, weak and irregular; sometimes slow, weak and intermittent.

Fever may be present during the prodromal stage, if any. The temperature is usually raised at the onset and during active vomiting. It reaches to 102-103° F., but may be up to 105° F. Hyperpyrexia has occurred at the termination of fatal cases (Langmead).

Generally there is great restlessness, but the mind is clear. Sometimes there is delirium. Fatal cases develop drowsiness and apathy, and die in convulsions or coma, like that of fatal diabetes.

The urine is scanty, hyperacid, contains acetone bodies, and may contain albumin and hyaline casts. Excess of xanthin bodies, deficient excretion of uric acid, indicanuria, hæmaturia, and suppression have all been noted at times. Acetonuria may be present from the onset, or not until the end of the attack. In Marcy's fatal case indican and acetone were absent in the prodromal stage, and present on the second day. Howland and Richards found much indicanuria, increased uric acid,



diminished sulphuric acid, and an increase in the unoxidised sulphur. Probably these results indicate increased tissue destruction and deficient oxidation.

*Course and Prognosis.*—Between the attacks of vomiting the child is generally quiet and sleeps. The ordinary duration of an attack is two or three days, and the temperature then becomes subnormal. Six hours is the shortest period. Attacks may be so severe and prolonged as to endanger life from cardiac asthenia. Nephritis is the most dangerous complication. Up to 1908 seven fatal cases had been recorded, four of which were examined after death, so the prognosis is distinctly favourable. Almost always the attack ends quite suddenly. The younger the child the greater is the prostration and the worse the prognosis. The attacks recur at irregular intervals, occasionally regularly every month, and cease about puberty. The intervals vary from a few weeks to a year, usually one to three months. Each attack may prove the last. In a few instances they have been replaced by migraine. As soon as the vomiting ceases health and strength are rapidly recovered, and between the attacks the child is quite well, except in so far that the younger children are unduly liable to gastric and intestinal troubles.

*Morbid Anatomy.*—The liver presents the characteristic fatty appearances seen in acidosis, to a variable extent. Fatty changes have been found in the kidneys and the heart. Langmead obtained a definite amount of acetone from the blood.

*Pathogenesis.*—The worst cases closely resemble the late stages of diabetes. It is almost certain that various types of recurrent vomiting have been grouped together under this title, and that some of them are due to a disorder of metabolism, such as occurs in acid intoxication and post-anæsthetic poisoning. On the other hand some may be hysterical, due to a neurosis, an explosion consequent on slight gastro-intestinal disturbance; and others associated with the arthritic diathesis. In favour of a primary nervous affection are the hereditary factors, the neurotic temperament of the child, the occurrence in brothers and sisters, the effects of excitement and emotion as exciting causes, the sudden onset and cessation, and the beneficial results of treatment by suggestion. Acetonuria in these cases may be gastro-intestinal in origin, or the effect and not the cause of the vomiting. The arthritic cases may depend on auto-toxins or intestinal toxins, related to or identical with purin bodies, which may account for those attacks subsequently replaced by migraine.

The most important and dangerous group includes those dependent in some way on hepatic inadequacy. Acetonuria occurs before the vomiting in some cases, so cannot always be due to it. The ordinary bilious attack, consequent on error in diet, is of this type. Perhaps in these cases we have to deal with a functional disorder of metabolism, in which the child does not ingest or is unable to metabolise a sufficiency of carbohydrate food,



with the result that profound nutritional disturbance, hepatic incompetence, acid intoxication and autolysis of liver cells are induced. Auto-intoxication is secondary to the inadequacy of liver function. The early stages are hardly those of acid intoxication, but the later ones may be partly or entirely due to it. As a rule there is no deficiency of food, so the defect must be in the oxidising powers of the liver and other organs. The fatal cases occur in fat children, and any check to oxidation will induce acid intoxication in a child predisposed to it by an excessive fatness or an undue quantity of fatty food. Poisoning by the products of intestinal putrefaction, indol and skatol, etc., may be an exciting factor in that they reduce the oxidising power of the tissue cells.

*Hepatic Inadequacy* gives rise to white or clay coloured stools, complete acholia without jaundice, and occasionally cystinuria. The excretion of uric acid is decreased and indicanuria increases as the patient gets worse. Sugar metabolism is unaffected. Sometimes the liver is swollen, and jaundice may be present towards the end. It may be the first stage of the attacks of recurrent vomiting with which it is at times associated.

Accurate *diagnosis* is essential to judicious treatment. First attacks are likely to be mistaken for ptomaine or other poisoning, acute indigestion, acute gastritis, gastric influenza, appendicitis, intussusception, intestinal obstruction, renal disease, and meningitis. Cases have even been operated on because of the severe vomiting and constipation. A rectal examination should be made. Later, on account of the emaciation, the affection simulates tuberculosis, Addison's disease, cerebral tumour, and diabetes. The vomit should be examined for excess of hydrochloric acid, and the urine for acetone bodies and indican. The acetone odour of the breath must be looked for. Recurrence of attacks and rapidity of recovery establish the diagnosis.

Further, it is important to try and differentiate the purely nervous cases from those dependent on hepatic inadequacy and associated with acid intoxication; and those in which the gouty and arthritic elements are marked. A typical case, in a girl of 6 years, in whom the attack occurred every 4-6 weeks and lasted 3 days, was partly neurotic. A sister had hysterical anorexia at the same age. Probably the neurosis was not the sole factor, for the attacks came on during sleep, with a temperature of 104° F. and marked constipation.

The *treatment* of the purely nervous cases consists in overfeeding, massage, a darkened room, purge, temporary starvation, valerianates and suggestion. In the acetonuric cases the measures advised in the treatment of acid intoxication must be adopted (p. 155). During the prodromal stage give a large enema; calomel gr.  $\frac{1}{4}$  and sod. bicarb. gr. v, half hourly for 10 doses, followed by a saline laxative in 2 or 3 hours; sodium benzoate gr. v every 3 hours, beginning 4-5 hours later; no food; water if it can be retained. Once the vomiting has begun nothing seems to control



it. Iced sugar solution, milk of magnesia, and sod. bicarb., citrate or lactate, may all be tried. After 24 hours starvation give small amounts of broth, albumin water, cereal decoctions, whey and peptonised milk. Morphia and atropin, chloral per rectum, strychnia and saline injections may be useful or necessary to tide over a crisis. In the intervals rely on a quiet, uneventful outdoor life; a liberal supply of water; a diet limited in fat, and consisting chiefly of milk, cocoa, vegetable soups, cereals, well-cooked fruits and vegetables, fish, chicken, and mutton. Attend to the digestion and bowels with rhubarb, soda, grey powder, calomel, and the sulphate and phosphate of soda. Salol and other intestinal antiseptics may be tried.

**Glycosuria. Diabetes.**—True diabetes is a very fatal disease in children, and fortunately uncommon. From 1-2 per cent. of all cases begin under 10 years of age. Bell (1896) reported a fatal case at the age of 3 months. It must not be confused with simple glycosuria, the transitory appearance of sugar in the urine, such as is seen in whooping cough, after anæsthesia, as the result of excessive starchy or saccharine diet, and even in pneumonia and diphtheria. Other copper-reducing bodies in the urine can be distinguished by their lack of fermentation power.

*Alimentary Glycosuria* is a defect in the metabolism and assimilation of carbohydrate foods. It is apt to occur in the children of diabetics. The mildest type is that in which sugar only appears in the urine after the ingestion of a large amount of grape sugar, more than 50 gms. In the more marked cases the sugar appears after an excess of starchy food. These children can be tested by a mixed dinner containing 8 oz. of carbohydrates, and examining the urine 2-4 hours later. They are liable to acne and obesity, and may develop prematurely.

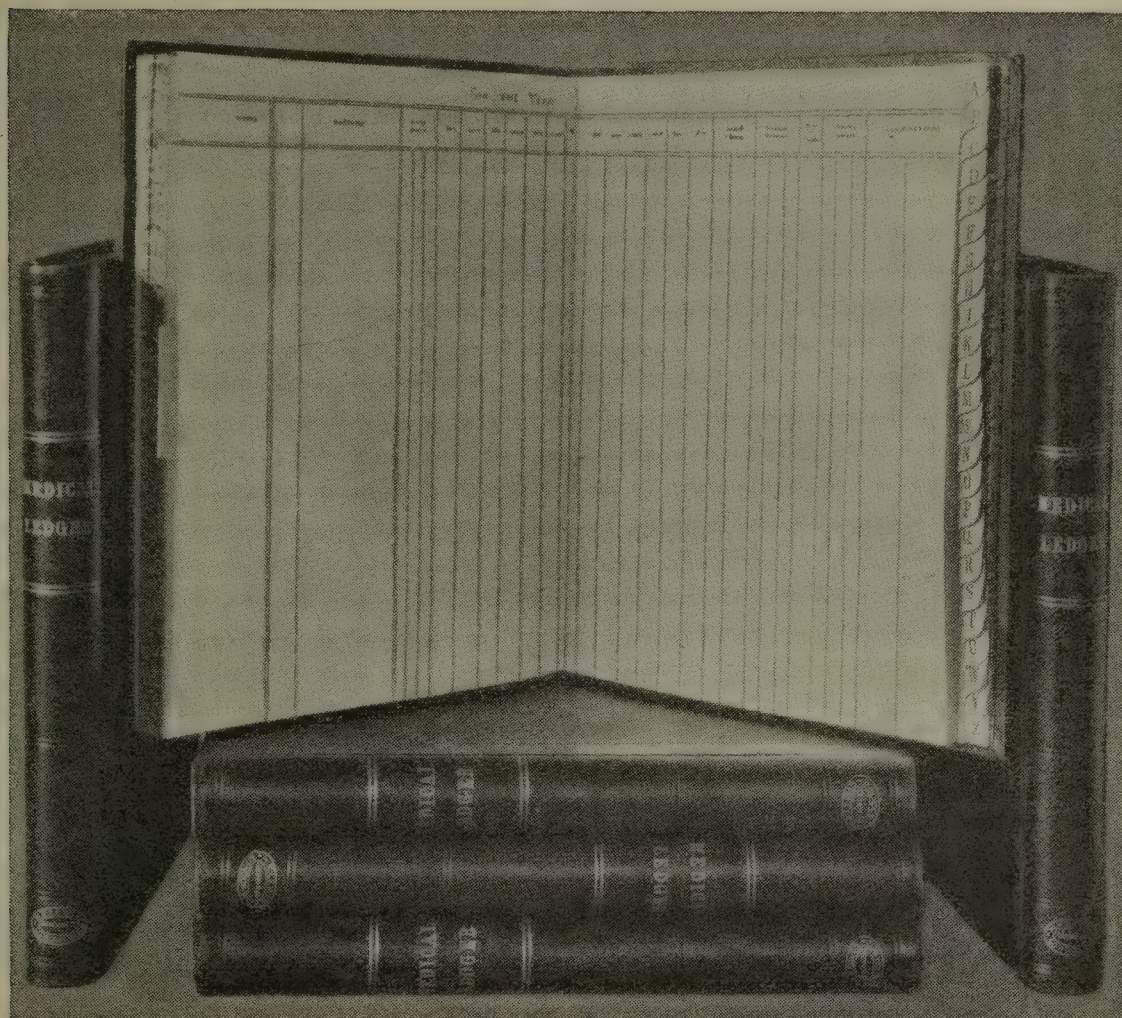
*True Diabetes* can be divided into two groups, with no strict line of demarcation. The mild cases exhibit a considerable and persistent amount of sugar, which disappears on the reduction or omission of carbohydrate food. The severe cases are little or not at all affected by diet. Possibly they are of pancreatic origin. Mild cases may become severe, and occasionally a simple glycosuria develops into a mild diabetes, rarely into a severe form.

A racial tendency is seen among Hindoos and Jews, but there is no special frequency among Jewish children. In about one-fifth there is a family history of the disease, and often a history of neuroses, gout or tuberculosis. Several children in a family may be affected at about the same age. Cases have been reported in the first year of life, and one is said to have been congenital. The yearly frequency is about the same in the first ten years of life, and then increases. Both sexes are equally affected. Frequently no cause can be found. Some cases have followed injury to the head, convulsions, operations, fright, over-exertion, rapid growth, dentition, influenza, typhoid and other fevers, sore throat, gastric catarrh, and severe burns. A few may be due to a pancreatic fibrosis, consequent



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on congenital syphilis. Extirpation of the pancreas only produces glycosuria in those birds which eat meat. Possibly the disease is dependent on decomposition of meat foods inducing hyperactivity of the thyroid (Lorand), especially if associated with degeneration of the Islands of Langerhans. An excess of meat and carbohydrates is more likely to cause it than if the meat is omitted.

*Symptomatology.*—Usually the first symptoms are wasting, thirst, polyuria, languor and joint pains. Enuresis may be the first sign. An excessive appetite is an uncommon feature, and there may be no obvious wasting. An insidious early stage may be overlooked, and end in death from coma within two days of the first suspicion of illness.

The amount of urine varies greatly, e.g., 50 oz. or less in a girl of 10 years; 110 oz. in a boy, aged 8, who looked quite well; up to 10 pints in a baby aged 15 months (Langstein); up to 10 quarts in a 10 year old boy (Wegeli). The specific gravity ranges from 1025-1050; the percentage of sugar from 5-10, and the daily amount up to 3000 grains. These amounts may be exceeded. In examining the urine take a mixed specimen from the total daily excretion, and not from the morning urine only, or an erroneous impression of the severity of the disease will be formed. Albuminuria is not infrequent, and is generally present shortly before death, with short thick granular and cylindrical casts. Acetone bodies are serious indications. Diacetic acid is of grave import, and may remain though sugar temporarily disappears. The ammonia excretion is increased. Purin excretion is also increased, even though on a purin-free diet.

All the signs of the disease in adults occasionally are seen in children, viz., harsh dry skin, furunculosis, carbuncles; acetone breath, raw beefy tongue, dryness of fauces, thirst, toothache, decayed teeth, abnormal appetite, constipation; balanitis, pruritus vulvæ; anæmia, pale and sunken face, small and frequent pulse, subnormal temperature, abdominal pain, vomiting, restlessness, dyspnœa, air-hunger, distress, heavy breathing, cyanosis, mental confusion, apathy, progressive coma, and occasionally convulsions. Complications are rare because of the rapid progress.

The *diagnosis* is simple, if the urine is examined and temporary glycosuria can be excluded. In infancy it may be mistaken for marasmus or tuberculosis, for thirst and polyuria are not always conspicuous. Lactosuria may be found in breast-fed infants. Transitory glycosuria should clear up in a few weeks.

*Course and Prognosis.*—The onset is more sudden and the course more rapid than in adults. The younger the child the worse is the prognosis. Few live beyond two years and the worst die within a few weeks, or even within a couple of days of the onset of acute symptoms, the disease being previously unsuspected. Six months is an average duration from the date of diagnosis. Mild cases which yield to dietetic treatment are possibly curable. Those cases are most favourable which can be thus



influenced, and have no acetonuria. They may live for 3-6 years. In one child glycosuria persisted for 8 months, then remained absent for several months, but returned and proved eventually fatal. Even if mild cases are doing well on a limited diet, the carbohydrate tolerance is liable to break down on any illness or indiscretion, and generally fails without such cause. Neither diet nor drugs produce much effect in severe cases. A few die from tuberculosis, pneumonia or other intercurrent disease. The common termination is that of coma, ushered in by drowsiness and air-hunger, and preceded by anorexia, nausea, vomiting, and abdominal pain. A sudden fall in the sugar excretion often indicates the onset of coma or of an acute illness.

*Treatment.*—Adopt careful hygienic precautions against chill. Avoid excitement and overwork, mental or physical. Allow prolonged rest in bed at night. Let the child live an ordinary life, as far as possible, with change of air and even sea-bathing. Regulate the diet on the usual lines, reducing the carbohydrates only if the acetone reaction of the urine is absent. Oatmeal and potato seem to be tolerated best. The oatmeal cure consists in a daily diet for adults of oatmeal 150 gms., butter 150-200, roborat 60-70, eggs 4-5, and some wine. It is given for a week or two, and slowly replaced by other foods. The general principle of dietetic treatment is to establish tolerance, to keep within the limits, and periodically to give a carbohydrate-free diet for several days.

For thirst rely on tepid drinks, water, skimmed milk, buttermilk, water with phosphoric acid or cream of tartar, lemonade made with 45 grs. of citric acid and 3 drs. of pure glycerine to the pint of water, alkalies, and alkaline mineral waters.

For coma give large doses of bicarbonate of soda, and even earlier to prevent its onset as soon as there are signs of acidosis. Guard against constipation by Apenta water, Carlsbad salts or Epsom salts. Measure daily the amount of urine, and estimate the quantity of sugar passed. The fermentation test is a sufficiently accurate guide, in conjunction with the weight of the patient, of the effect of the diet on the excretion of sugar and the general health. Stimulants are often useful. Of drugs the chief ones recommended are codeine, opium, atropine, bromide of arsenic, liquid extract of ergot, aspirin and salicylates.

*Scurvy.*—Scurvy is rarely seen in this country, except in infants brought up on cooked milk or a proprietary food for a long period of time. It was recognised and described by Glisson (1651) as distinct from rickets and having no essential connection with it. Confusion has arisen because it has been called at different times, *acute*, *scurvy*, or *hæmorrhagic rickets*. These names should be given up. Cases have been described by Sir Thomas Smith (1876) as “*hæmorrhagic periostitis*”; by Gee (1881) as “*periosteal cachexia*”; by Cheadle (1878) as “*scurvy supervening on rickets*,” and (1882) as “*osteal or periosteal cachexia and scurvy*,” recognising that



the affection was true scurvy. As the result of a full description by Sir Thomas Barlow (1883), who showed that the scurvy was the central factor, and the rickets variable and unimportant, it has been known as *Barlow's Disease*. Subperiosteal hæmorrhage is not common in the scurvy of adults. In infants the actively growing tissue is very vascular and, therefore, more liable to hæmorrhage.

It is by no means uncommon, though often unrecognised, in infants. The mildest cases may be entirely overlooked, the child recovering by reason of some change in the diet. An individual susceptibility may show itself in twins, one only being affected. Three-fourths of the cases occur between 6 and 12 months of age. It is rare under 6 months and after 15 months of age. A few cases have been recorded in breast-fed infants, even as early as the fourth or fifth week of life, but they are open to criticism. The disease occurs rather more often in boys, in fat anæmic babies and in the ill-nourished, at any period of the year, in town or country, and in any climate. Possibly plenty of fresh air may postpone the onset. It occurs in every grade of the social scale, but more frequently in private than in hospital practice, for the children of the poor are rarely brought up for a sufficiently long period on cooked milk and often receive tastes of various antiscorbutic foods. The following table shows the diet of a large number of collected cases :—

Breast Milk	..	..	..	21
Breast Milk with additional food	..	..	2	
Uncooked Cow's Milk	..	..	5	
Pasteurised Milk	..	..	40	
Boiled Milk	..	..	14	
Sterilised Milk	..	..	122	
Condensed Milks	..	..	72	
Proprietary Foods	..	..	250	
Dried Milk Foods with water only	..	..	21	

Some of the proprietary foods produced the disease although given with fresh milk. Long continuance of the particular diet is an important factor. Thus, a mild case developed in a child, aged 10 months, who for 8 months had been fed upon a mixture of cream, milk, sugar and water, sterilised for 20 minutes. Another child, aged 15 months, had taken pasteurised milk only for a year.

*Pathology.*—A change is produced in milk by heat, reducing or destroying its antiscorbutic property. It is no longer a fresh food. The greater the heat and the longer the duration of heating, the greater is this destruction. For this reason condensed milks and patent foods are particularly injurious. Possibly it is connected with the destruction of enzymes; changes in the protein; or the conversion of soluble salts of lime into insoluble forms, e.g., the formation of insoluble calcium phosphate,



and of the crystalline and less soluble calcium citrate from the more soluble amorphous salt (*Citric Acid Theory*). Nevertheless, the addition of citrates to the food, or of alkaline phosphates to keep the citrate in solution, has apparently no remedial action. Nor is it due to the absence of potash salts in organic form, for these salts are not curative. Cooking does not affect the antiscorbutic property of potatoes and greens. The theory that it is due to tainted food or ptomaines (*Tainted Food Theory*) is certainly not supported by evidence derived from cases in infancy; for it arises occasionally in the breast-fed and on diets in which neither animal food nor microbes are present; and there is no evidence of ptomaine poisoning. Sir Almroth Wright ascribes it to an *Acid Intoxication*, very similar to that produced experimentally in herbivora by the ingestion of a surplus of mineral acids over bases. Thus, a diet of meat and cereals with the exclusion of green vegetables and fruits will produce it. Meat and cereals contain a large excess of mineral acids, especially phosphoric acid, while vegetables and fruits contain an excess of bases. Wright divides foods into: (1) Acid or scorbutic, viz., eggs, fresh and salt meat, and many cereals; (2) Neutral or non-scorbutic, viz., sugar, vegetable oils, and animal fats; (3) Alkaline or antiscorbutic, viz., milk, blood, lime-juice, orange juice, potatoes, green vegetables, etc. In adults he found the alkalinity of the blood was decreased, and he advises the administration of the salts of the oxidizable organic acids, e.g., sodium lactate, to restore the normal blood alkalinity. But the reduced alkalinity, if present, is due to anæmia. Moreover, scurvy does not occur in Acidosis, and lime-juice raises the acidity of the urine, yet cures the disease. According to Hutchison, Wright's division of food-stuffs is based on inaccurate chemistry, and only eggs and meat, deprived of all blood, leave an acid ash. Recent experiments by Axel Holst of Christiania (1908) upset Wright's theory. Scurvy was produced in guinea pigs by a diet of bread and dry cereals, but not if fresh raw carrot was added; on a diet of compressed dried potato and water, but not if cabbage juice was added; on a diet of dried carrot, even if soaked in a solution of bicarbonate of soda; and on a diet of dried barley and water, but not if the barley was allowed to sprout first. Yet in the sprouted and unsprouted barley the same amounts of acid and salts were present. Nor did the addition of sodium citrate and calcium carbonate to these diets prevent the disease. It may, therefore, be accepted that scurvy is not due to acid intoxication or reduced alkalinity of the blood. All that we can say at present is that it is the result of the lack of some property in fresh food, which is destroyed by heat, or an inadequate supply of fresh food, and that it may be accentuated by unsuitable diet containing an excess of starch.

*Symptoms and Course.*—The onset is gradual, with fretfulness, impaired appetite, malaise, anæmia and loss of weight. Sometimes immobility of a limb is the first sign to attract attention, or some general tenderness, the



child crying when handled or even approached, for fear of being moved. There may be a history of a previous attack, lasting for a week or two and ending in recovery. If so, it will be found that the actual cause was not recognised, and the improvement was due to some temporary change in the diet. Not infrequently hæmaturia is the first sign. In the mildest cases, although some of the teeth are cut, the gums may be unaffected or exhibit swelling, hyperæmia and slight purplish discolouration. They show no change unless the teeth are cut or almost through. In severe cases they are swollen, bulbous, ecchymotic, varying in colour from dusky to purple, and may ulcerate and bleed freely (hæmorrhagic gingivitis). The swelling may form a fungating mass so great as to hide the teeth, sloughs form, and the breath becomes very offensive. Sometimes there is hæmorrhagic discolouration of the hard palate. The tenderness and pain cause complete immobility of the limbs, especially the legs, with extension, eversion, and pseudo-paralysis; or the legs may be kept partially flexed and rigid. This is due to local pathological changes, not necessarily causing a visible or palpable swelling. Generally on examination there are found one or more colourless, painful, tender swellings, often symmetrical. The most common site is the lower third of the thigh, and the least common is the upper limb; occasionally the clavicles, scapulæ and ribs. The swelling is due to subperiosteal effusion of blood, taking place over the long bones, and sometimes causing separation of the epiphysis and soft crepitus. The superjacent skin is smooth, tense, shiny, sometimes bluish, not hot, and rarely pits of pressure. Hæmorrhages may occur over the flat bones of the skull, forming large bluish swellings, like sarcoma. The thorax is often tender. The sternum may sink in from fracture or separation at the costo-chondral junction, and cause a peculiar flattening of the front of the chest. Bleeding often takes place from the nose, and occasionally from the ear, into the eyelid, behind the eyeball, from any mucous surface, into the muscles, under the skin, and into various serous cavities, the cranial cavity, joints, and any organ of the body. Anæmia is independent of hæmorrhage and often marked; the blood is deficient in red cells and percentage of hæmoglobin. Muscular pains are severe and cause loss of rest; fainting attacks are not uncommon; occasionally the long bones get broken. Fever is due to complications, or to disturbed sleep and general malaise from pain. In older children similar symptoms occur, but the disease as a whole approximates more closely to the adult type. It is generally due to deficiency of vegetable and meat foods.

*Morbid Anatomy.*—The bone symptoms are due to extravasation between the actively growing periosteum and the diaphysis, leaving the bone bare and impairing its nutrition. The periosteum may be completely separated, the epiphyses detached, and the shaft free in a cavity containing blood and clot. Bone forms in the detached periosteum, for its osteogenetic power is not destroyed. Lymphoid cells in the marrow,



at the junction of the cartilage and shaft of long bones, are replaced by a reticular tissue, poor in cells and often the seat of hæmorrhages. The changes in the bone marrow and the destruction of osteoblasts lead to inefficient formation of new bone. Consequently the normal bone absorption causes thinning of the bone in these regions, rarefaction, partial dislocation at the epiphyseal line and fractures, partial or complete, from very slight violence. There is no severe displacement for the periosteum is untorn. It is rare for necrosis of the bone to take place.

*Complications* are such as are due to hæmorrhages in various situations, rather to be regarded as symptoms, and those due to associated rickets. Intrarenal hæmorrhage may give rise to blood, albumin and casts in the urine, and may be mistaken for acute nephritis. Albuminuria may occur alone. Acute nephritis is rare. Diarrhœa is a serious complication; pyelitis prolongs the illness; broncho-pneumonia may prove fatal. Feeding may be difficult on account of the condition of the gums.

*Diagnosis* is only difficult in mild cases in very young infants before any teeth are cut. The disease is often unsuspected for the child may be very fat, sometimes abnormally so, and the suspicion of scurvy not aroused. The fatness also renders it difficult to recognise a moderate subperiosteal swelling, especially if it is symmetrical on the two sides. The child may have been brought up amid most excellent surroundings. The cardinal signs are anæmia pain and tenderness, all of which commonly occur before the swelling of the gums. Fretfulness and spongy gums, or hæmaturia, may be the only symptoms. Whenever an infant suffers from pain and tenderness in the limbs, it should be carefully examined for scurvy and inquiry made into the diet. The affection is often mistaken for rickets, when rickets is present, but it is doubtful whether this disease is ever painful. The pseudo-paralysis of the lower limbs in a rachitic child with marked kyphosis may be erroneously diagnosed as pressure paraplegia due to spinal caries. The rarity of rheumatism at this age ought to prevent such a diagnosis being made. A swelling limited to one limb or epiphysis has been mistaken for sarcoma, acute periostitis or epiphysitis. Syphilitic epiphysitis occurs in wasted infants under six months of age. Multiple osteomyelitis is extremely rare and, like localised osteomyelitis, is accompanied by high fever. Epistaxis may be regarded as accidental; renal hæmorrhage ascribed to sarcoma; subcutaneous hæmorrhages mistaken for purpura; and the condition of the gums put down to acute leukæmia. If there is no swelling and the gums are unaffected, the pseudo-paralysis can be distinguished from that of infantile palsy by the tenderness and pain. Orbital hæmorrhage, usually in the upper lid, is very suggestive. In only one case have I found marked tenderness and dusky gums unassociated with anæmia. Ulcerative stomatitis is differentiated by its not being limited to the gums.



*Prognosis.*—Mild cases get rapidly well, and show definite improvement as regards the tenderness in a day or two, if put on appropriate treatment. In bad cases the prognosis must be guarded because of the liability to severe hæmorrhage, syncope from the profound anæmia, and death from diarrhœa and exhaustion. Convalescence is prolonged; absorption of hæmorrhage and thickening slow; the separated periosteum may form a bony sheath. Treatment may fail in advanced cases of anæmia, with extreme cachexia, and enteritis or broncho-pneumonia.

*Treatment.*—Rapid recovery on an antiscorbutic diet is strong confirmatory evidence of the accuracy of the diagnosis. Put the child on a diet of uncooked milk and the juice of oranges or grapes, if it is under six months of age, with the addition of cream and meat juice if necessary. Over this age add potato cream to the diet; it is made by adding 2 teaspoonfuls of the outer part of a baked floury potato to 1 oz. of milk, and from 1-2 teaspoonfuls is given alone, or in the first part of the milk feed, 3 or 4 times a day. If given to very young infants, or for long, it is liable to upset the digestion. In mild cases the milk need not be given fresh. It can be just brought to a boil, mixed with fresh barley water, and the juice of fruit, vegetables or meat added, or given independently. Older infants may have vegetable soups or purées made with carrots or potatoes. The child should be handled, moved, and dressed as little as possible; affected legs placed between sandbags, and an affected arm bandaged gently to the side. In rare instances extreme periosteal swelling must be relieved by a small incision and packing. The gums can be painted with an antiseptic astringent lotion of tincture of myrrh and Listerine. Drugs are unnecessary. Sodium lactate can be given and is harmless. Iron, arsenic, and cod-liver oil are useful in convalescence.



## CHAPTER XIV.

### INFANTILE ATROPHY.

*Syn. : Athrepsia (Parrot, 1877)—Malnutrition—Marasmus—Inanition.*

The Registrar-General divides wasting diseases into those due to premature birth, congenital defects, injury at birth, atelectasis and atrophy. It is a curious and inexplicable fact that the mortality is much greater among males than females. The total mortality rate for wasting diseases has increased during the last thirty years, although there is a considerable decrease in the number of cases ascribed to atrophy alone. This may be the result of stricter methods of registration and more accurate diagnosis of the causes of atrophy. Many stillborn infants formerly escaped registration, but now appear in statistics as prematurely born. Nevertheless, it seems that there is an increase in the inherent predisposition to death in early life from defective vitality, congenital defects, and similar causes.

*Physical Deterioration.*—There is little evidence that the race as a whole is progressively degenerating. Without doubt a very large number of imperfectly developed children and adults are in existence. Relative statistics of age, weight and height in different decades, are of comparatively little value, for the proportion of the unfit or badly grown is large and tends to increase yearly. This is the natural result of improved hygiene, sanitation, cheap food, and medical science. Many infants, who in former days succumbed to the stress of existence, now survive, grow up, and propagate the species. A great amount of good can be done by directing attention to, and counteracting as far as possible, those causes which prevent proper growth and development without actually causing deterioration.

Imperfect development depends upon ante-natal conditions, diet, and environment. About 80 per cent. of all children are born healthy. The debility of the remainder is due to morbid heredity or conditions affecting the mother during pregnancy. The main hereditary factors are tuberculosis, syphilis, alcoholism, neuroses and general debility. The influence of morbid heredity tends to die out by extinction of the family, or is corrected by marriage with healthy subjects. The factors affecting the mother during pregnancy are referred to in a previous chapter (p. 21).



Anything that impairs the health of the mother is more or less injurious to the child. After birth development is mainly a question of suitable diet. Other factors being equal, breast-fed infants are stronger, healthier and less prone to disease than those artificially fed. Hebrew children are better grown, heavier, less rachitic, and have sounder teeth than those of the Gentiles. This is due to the fact that the Jewish mother is better fed and cared for during pregnancy, and that her child is almost always breast-fed during infancy, and gets a more suitable diet during early childhood.

The advantages of cheap food have been rather overrated. The craze for cheapness has led to the adulteration of food and the provision of cheap, tinned, savoury and unwholesome foods, which form an injudicious and unsuitable diet for young children. It is by no means uncommon to find a child fed on condensed milk, proprietary foods, tinned meats and fish, sausages, and pickles; tasty articles of diet which deprave the appetite for simpler food, and give rise to gastro-enteritis and malnutrition, and predispose to tuberculous infection. Tea, alcohol and smoking in early life are also distinctly harmful; so, too, the use of drugs, such as opiates and white bryony, and patent medicines.

Other deleterious factors are overcrowding, bad hygiene, insufficient clothing, exposure to inclement weather, insufficient sleep, over-fatigue, and general neglect. Education is by no means an unmixed blessing. Children, who in former days added to the family exchequer, are kept at school, and the parents have difficulty in providing for them. Many of them suffer from over-pressure and, in winter especially, from insufficient fresh air, light and exercise.

The general effects of these various causes are the production of undersized, weakly adults. They are small, stunted, and have bad digestion, which is further impaired by indulgence of the craving for tasty and stimulating foods and drink. Little prospect is there of such people becoming parents of strong and healthy children. Morally, physically and mentally they are ill-developed, and their children are likely to be congenitally feeble and liable to atrophy.

**Atrophy.**—Although malnutrition, atrophy, wasting and marasmus are merely symptoms or names for various degrees of wasting, there is a general condition deserving the name of *Infantile Atrophy*. It must not be confounded with simple wasting and inanition due to starvation, though it can be produced by insufficient or unsuitable diet. Malnutrition indicates that constructive metabolism is not greater than destructive. It varies in degree and rapidity of progress. It may occur at any age, but is a most marked feature during the first year.

It can be ascribed to defective diet, inability to suck, indigestion, bad hygiene and neglect, an inherited defect of tissue, an inherited taint, prematurity, defect in the digestive organs, malformation and organic disease.



The dietetic factors are over-feeding, food insufficient in quality or quantity, an unsuitable breast-milk or artificial mixture, bacterial contamination, and failure of assimilation.

Inability to suck or swallow may depend upon structural defects or organic disease, such as hare-lip, cleft palate, large tonsils, palatal palsy, or inflammatory affections of the mouth and throat. It may depend upon nasal obstruction, from snuffles or adenoids; upon small and retracted nipples or an unsuitable teat; on complete anorexia or unsatisfactory food. Occasionally there is no obvious explanation, and in such instances it has been ascribed to imperfect co-ordination or development of the nerve centres for suction or deglutition. Sometimes such infants will begin suddenly to suckle normally. Pinard reported a remarkable case in which the reflex area for suckling receded gradually from the tip to the base of the tongue. The child died from hydrocephalus. In other infants the refusal of food depends on mental defect or on temper. If the use of the bottle has been unduly prolonged, the child may refuse semi-solid food.

Of the general causes most importance must be attached to prematurity and a family history of tuberculosis or syphilis. Even apart from actual disease there results a feeble vitality, due to the inheritance of imperfect tissues which are incapable of efficiently carrying out the functions of digestion, absorption or assimilation. By far the most important factor is the diet of the child, either alone or in conjunction with other causes. Hence arise the intestinal troubles, e.g., persistent indigestion, which are the chief cause of atrophy.

*Morbid Anatomy.*—Post mortem examination reveals sometimes congestion or oedema of the meninges and brain, hypostatic congestion of the lungs, collapse and pleural ecchymoses, and, rarely, sinus thrombosis. The gastric and intestinal mucosa is pale, sometimes swollen, and may show patches of congestion, erosions and sub-epithelial hæmorrhage. The kidneys are almost always pale; the glomeruli injected, cortex swollen, pyramids hyperæmic; with cloudy swelling and fatty degeneration of the epithelium, especially in the convoluted tubes. In prolonged cases the liver shows fatty and parenchymatous degeneration. The organs are generally pale; the lymph nodes, follicles and Peyer's patches may be a little enlarged.

*Pathology.*—The anatomical changes are the effect, not the cause of the wasting. The atrophy of the intestinal mucosa, to which the wasting has been ascribed, is part of the general wasting; while the microscopical appearances are due to gaseous distension of the gut and post mortem changes. In other words the disorder is one of functional impairment of digestion, absorption or assimilation. In those instances in which a sufficient and nutritious diet is apparently digested, and yet the child wastes, the functions of assimilation are in abeyance, defective or destroyed. With increasing feebleness the circulation becomes weaker, the lung



expansion imperfect, and oxidation incomplete. On insufficient evidence the condition has been ascribed to acid-intoxication ; to auto-intoxication from decomposing food ; to deficient activity of nutritive ferments, due to intestinal infection ; to defect in Paneth's cells, found in Lieberkuhn's crypts ; and to sub-acute or chronic infection of the intestinal tract.

*Symptoms* vary according to the age, the rapidity of the wasting and its duration. The most typical cases are seen in infancy. After the first year acute inanition and marasmus are less frequent, though they result from acute illness and from causes similar to those acting during the first year of life.

*Acute Inanition* or *Acute Atrophy* is characterised by rapid loss of weight, and is almost invariably associated with a deficient supply or an excessive loss of fluid. It may be due to unsuspected starvation. The child is pallid and prostrated. The extremities are cold, and the skin dry or, occasionally, covered with clammy sweat. The pulse is weak and frequent, breathing shallow and irregular, and temperature subnormal. The fontanelle is sunken, sutures overlapping, pupils small or pin-point, and the mental condition one of irritability or fretfulness, passing into a state of semi-stupor. The appetite is lost. Vomiting may be absent, or occur after several feeds have been taken, because of distension of the stomach due to loss of its motor power. The stools are sometimes normal, but usually contain undigested food, and frequently mucus. If no food is taken, they consist of epithelial debris, mucus and bile, and look like meconium. The asthenia increases. It may be associated with diarrhoea and slight cyanosis of the lips, and prove fatal in a few days. The younger the infant, the more rapid is the progress. In less severe cases life may be prolonged from three to six weeks.

An infant is said to be in a state of *Malnutrition* when it is much below its proper weight, remains stationary in weight, or gains very slowly for some considerable time. In addition there is a deficiency in the other vital processes of development, such as growth in length, acquirement of the functions of sitting, standing and walking, and mental development. The muscles are flabby, the ligaments weak, and the joints lax and insufficiently supported. The fontanelle is depressed. Dentition may be delayed, but it is often accomplished without trouble or discomfort. Such infants are more or less anæmic, sharp-featured and emaciated, with cold extremities, dark rings under the eyes, deficient elasticity of the skin, and a temperature which readily sinks below normal. They are fretful, irritable, restless and sleep badly. Especially are they subject to gastrointestinal and pulmonary troubles, and frequently die from an attack of bronchitis. The appetite is poor or capricious. Indigestion is readily set up and attacks of diarrhoea are frequent. Sometimes there is an actual catarrhal enteritis or entero-colitis.



As the process continues it passes into the more serious stage of wasting, to which the name of *Marasmus* has been given. It must be clearly understood that marasmus is not a disease, but is merely a late stage in the course of malnutrition from various causes. It is consequently common during the first year of life, not infrequent in the second year, and may occur at later ages as a sequel of prolonged wasting diseases, such as persistent vomiting, tuberculous affections and cirrhosis of the liver.

In infancy the general history is that the child has been always delicate or that it was apparently healthy at birth and while breast-fed; that it began to waste when it was weaned; that the wasting has steadily persisted in spite of various methods of feeding, tried by the mother, recommended by the neighbours, or suggested by specious advertisements; and that in spite of all treatment the baby has got thinner and thinner, and has had frequent attacks of vomiting and diarrhœa.

In a typical case the facial aspect is that of old age. The skin of the face is wrinkled, thrown into folds on the forehead and between the eyes, has lost its elasticity and is devoid of subcutaneous fat. All over the body and limbs it is lax and easily raised from the subcutaneous tissues. It is harsh, dry, and hangs in folds. The bones stand out sharply, giving the features a pinched look; the eyes and temples are sunken, and the pupils small; the chest and extremities show little more than skin and bone; the abdomen is protuberant and tympanitic; and the terminal phalanges bluish. The temperature is low, and may fall below 95° F., even to 90° F. The blood is deficient in hæmoglobin; the circulation feeble; and the face pallid, earthy or ashen in colour. The tongue is red and dry, and both it and the mouth are often covered with patches of aphthæ. The appetite may be completely lost or abnormal, and the state of the stomach and bowels as in malnutrition. Even water may be vomited, and if the stools are apparently well digested they are unduly large for the amount of food taken. The mental condition is one of fretfulness and irritability, or of listlessness and apathy.

As the child gets worse, it is very liable to develop œdema of the dorsum of the feet and hands, and later of the eyelids, face and rest of the body. Effusions into serous cavities are absent. Diarrhœa may become less or cease. Petechial hæmorrhages are apt to appear on the lower part of the abdomen, the whole abdomen, or on the trunk, upper part of the thighs, and elsewhere.

In simple malnutrition and acute inanition complications are usually absent. Aphthous stomatitis may occur at any stage of wasting, and helps to maintain or increase the malnutrition. Herpetic stomatitis is less frequent, but is liable to occur in repeated attacks in chronic cases. Temporary, mild or severe, enteritis or entero-colitis is common. In prolonged wasting redness of the skin appears over the sacrum, heels, occiput, and other bony prominences, and may proceed to the formation of



bed-sores. Erythema and superficial ulceration of the buttocks arise from irritant discharges. Pustular boil-like spots appear on the scalp, and similar boils or subcutaneous abscesses on the trunk and limbs. The glands are susceptible to enlargement, and may reach the size of large peas or small nuts in the neck, groins and axillæ. Reflex opisthotonos is not uncommon, and the babe may lie continually with its head thrown back, spine bent and heels drawn up. Vomiting and enteritis are serious complications.

Improvement is indicated by gain in weight, provided there is no oedema, better appetite and digestion, less fretfulness, and a temperature nearer the normal level. The downward course is usually gradual, and death results from asthenia, sometimes in convulsions. It may be preceded by a period of unconsciousness of more or less sudden onset. It may take place quite suddenly, although no change has been noted in the state of the child; due to cardiac syncope, perhaps the result of flatulent distension of the stomach. Quite sudden collapse may also be induced by pulmonary collapse, broncho-pneumonia, vomiting, diarrhœa or fits.

*Diagnosis.*—Acute inanition is usually recognised easily when due to insufficient food, if the possible cause is suspected. A baby, seen on the thirteenth day of life, was dying from starvation. It had lost 2 lbs. in weight and its stools contained no fæcal matter. The mother's breasts were large and fat, and the milk supply had been supposed to be efficient. On extra food the child rapidly recovered. The subnormal temperature generally enables acute diseases to be excluded, and the rapid onset of the wasting distinguishes it from that of marasmus, except when it occurs as a termination of chronic wasting.

The differentiation between malnutrition and marasmus in infancy is merely one of degree. Before making a diagnosis of simple malnutrition, care must be taken to exclude the common causes of wasting in which there is organic or constitutional disease, such as pyloric stenosis and other malformations of the alimentary tract, gastric and intestinal catarrh, pulmonary troubles, empyema, morbus cordis, rickets, congenital syphilis, and tuberculosis. Time after time one sees infants in whom tuberculous disease is suspected, and no evidence thereof found after death. Less often, at the autopsy on a "waster," extensive tuberculosis is found, although there has been no suspicion thereof during life and the temperature has remained continuously subnormal.

Methods of diagnosis in infancy include regular weighing; examination of the breast-milk or the artificial food; the passage of a stomach pump and examination of the contents of the stomach for mucus, hydrochloric and organic acids, and bacteria, and estimation of its motility; examination of the fæces; examination of the urine for albumin and indican, and less often of the blood and cerebro-spinal fluid.



In older children the suspicion of tuberculosis is very difficult to get rid of. The history, diet and mode of life must be carefully investigated, and the child examined thoroughly for evidence of lung disease and glandular enlargement in the neck, chest or abdomen. Chronic dyspepsia at the time of the second dentition is often a cause of marked wasting, with cough, anorexia, and sometimes headache and restlessness. It is gastric or intestinal in origin, and may be associated with pharyngitis, enlarged tonsils and adenoids. Do not forget that as the child grows in height it commonly loses weight, but that malnutrition associated with rapid growth is not always due to growth. Lack of development may also result from naso-pharyngeal obstruction or deficiency of thyroid secretion.

*Treatment.*—The prophylaxis of infantile atrophy includes all that is conducive to healthy growth and development. In treating a wasted infant the diet should be that appropriate for an infant younger in age, and the intervals between the meals shorter. For infants under 4 months a wet nurse is advisable. The diet should be weak in quality, and altered in accordance with the progress of the case. Changes must be made with extreme care. A slight excess of food may bring on acute indigestion or diarrhoea, and undo in 24 hours the good of weeks of careful treatment or even prove fatal. Breast-feeding may be prolonged, entirely or partially, up to the age of 18 months if the child is small and like a much younger infant. If it is not available, or is inadvisable, reliance must be placed on whey, cream and whey, peptonised and modified milk, condensed milk, albumin water, various malted and partly malted foods, and Benger's food. At first cow's milk must be much diluted and given very cautiously. The digestive capacity, not the age or weight, is to be taken as the guide to the diet selected. In mild cases the ordinary principles of feeding, suitable for a rather younger infant, can be adopted. In more severe ones the yolk of egg, bone marrow, raw meat juice, and cod-liver oil may be given, bearing in mind that marasmic infants digest fat with difficulty and may get diarrhoea. A suitable mixture, consisting of the yolk of one egg, glycerine 1 oz., olive oil or cod-liver oil 1 oz., and creosote 10 m., in doses up to 1 dr., can be given 4 times a day. In very severe cases the infant must be kept warm, rubbed with oil, wrapped in cotton wool, and surrounded with hot bottles, or put in an incubator.

The general measures suitable for all cases, modified according to the age, include a liberal supply of fresh air; general friction or massage with olive oil or cocoa butter; regularity in diet, sleep and bathing; and sponging with cold water, about 60-70° F., after the morning bath. If these cases are accumulated in institutions, ventilation must be free and each child allowed 800 c.f. air space. Alcohol, saline injection and gavage are sometimes needed.

No cases exercise the skill of the physician to a greater extent than some of these wasters. The longer the duration of wasting, the greater



is the care required and the more tedious is the cure. Much depends on the faithful carrying out of instructions, which should invariably be written down fully and clearly. The parents must have faith in the physician to whose charge they entrust the treatment, and above all must realise that the progress will be very slow. Experiments in diet are often fatal, especially in hot weather, and frequently bad results are due to changes in the food ordered by competent physicians successively called in consultation. No case is hopeless and no pains are wasted in trying to bring about a better state of health, for these babies ultimately become strong and healthy, if the atrophy is due to dietetic causes and not of constitutional origin. The utmost care is necessary, and plenty of fluid given. Water can do no harm. The stools must be examined daily and the child weighed twice a week or daily in the worst cases.

In older children the treatment consists of simple diet, regular meals, regular habits, and open-air life, cold sponging, limited studies, and no excitement. Forced feeding is inadvisable. Iron, arsenic and cod-liver oil are the best drugs, if the alimentary tract is in a satisfactory condition.

In every case of simple atrophy the prognosis improves with the age of the child, and varies directly as the care devoted to its treatment and its environment. It is worst in those instances of inherited constitutional delicacy, and is increased in gravity by hot weather. The mortality is high in hospitals, because only the worst and most prolonged cases are admitted, and the power of assimilation in many of them is apparently completely lost.



## CHAPTER XV.

### RICKETS.

Rickets was described by Glisson (1681), who gave it the name of "*Rachitis*," from a Greek word for the spine, on account of the deformity of the spine so commonly present and its similarity to the popular name "*Rickets*," which was probably a corruption of the Norman-French word "*Riquets*" for deformities. It is a common chronic disease of malnutrition, occurring early in life from faulty diet. Its increasing prevalence is due to the increasing inability of women to nurse their children and to aggregation in industrial centres. It affects the whole system, but the most evident effects are produced on the cartilages and bones. The osteogenetic tissues undergo abnormal proliferation and either lose or have never possessed the power of making good bone. The general disturbance of metabolism involves the nervous system, mucous membranes, muscles, and ligaments. The disease is not fatal in itself, but predisposes to illness which may be fatal, or would not be serious apart from the effects of the rachitic process. It is a grave source of alimentary and respiratory affections in infancy, of nervous phenomena less directly, and of deformities which may affect the individual throughout life and in the future mother prove a source of disaster during confinement.

Some cases are much more acute than others, and are associated with visceral lesions, profuse sweating and, possibly, some bony tenderness. In others the bone symptoms and resulting deformities predominate. In a third type the catarrhal symptoms are the chief feature. In a fourth variety, known as the "*Acrobatic*" type, there is a remarkable laxity of ligaments and muscles.

*Etiology.*—It exists everywhere, although it has been called "*The English Disease*." Climate and confined life encourage its production. It is common in the temperate zone; rare in the tropics, China, Japan, and the southern parts of Italy and Spain; almost unknown near the equator, infrequent in Iceland, Greenland, Norway, and Denmark. It is rare at high altitudes; more common in towns than country districts; rare among negroes; almost as common among the rich as the poor, though in a less severe form. The worst cases are seen in the spring, as the result of the climatic conditions and confined life of an English winter. Yet they do not occur in some more northern countries where winter is longer and



the child more indoors. If slight beading of the ribs or craniotabes be regarded as sufficient evidence of the disease, it will be found in 50 per cent. of all children living in towns.

The disease is not transmitted from the parents. Hausen states that a stallion begat seven rachitic foals, and that two of the mares subsequently had healthy foals by other sires. Possibly the mother's diet during pregnancy, if deficient in protein and fat, and deterioration of her health from any cause, such as frequent pregnancies, may act as predisposing and exciting causes, through the child being born delicate and with less resisting power, and the maternal milk supply being of poor quality. Sex has no influence. It begins at some period of the first dentition, does not often become evident before the age of 6 months—a fact suggestive of post-natal causes—and by the eighteenth month is distinctly marked; usually it begins at the age of 6-12 months. Intra-uterine cases have been reported, and a few in later childhood. It may occur in breast-fed infants because of a deficiency in the mother's milk. In bottle-fed infants it results from a diet deficient in fat, still more if it is also deficient in protein, and even more if carbohydrate is in excess. The food may contain the requisite percentages of the different constituents, but the assimilative powers of the child be impaired by its unsuitable character. It is very unusual to find a baby brought up on condensed milk, alone or in combination with a proprietary food, who is not definitely rachitic; these foods are deficient in protein and fat. The breast-fed infant may develop the disease and the bottle-fed one remain healthy. In the breast-fed, however, it is generally of a mild type. The importance of food must not be overrated; some infants will thrive upon almost anything, and not develop rickets.

Puppies fed by Guérin on a meat diet for 4-5 months became rachitic while others of the same litter, suckled, showed no sign of the disease. Bland Sutton found that young lions at the Zoological Gardens became very rachitic, if weaned early and fed on raw meat only. The addition of milk and cod-liver oil to the diet cured them in 3 months. Two young monkeys fed on a vegetable diet developed rickets. On the other hand similar feeding experiments on young animals have produced profound marasmus and death without causing rickets. These experiments show that an improper diet will produce the disease, though it fails to show itself under conditions which produce marasmus. The defect in the diet is a deficiency in fat.

Recent experiments by Findlay (1908) show that confinement and lack of exercise have an important bearing on rickets; so much so that he ascribes the disease to lack of exercise. Oxidation is diminished or carbonic acid excretion defective. He found that experimental animals and birds kept in cages often developed rickets, although they got sun and fresh air; and he induced it in puppies by confinement only, and could not do so by any diet if free exercise was allowed. In support of his views



it may be urged that the disease is common in towns and the winter months ; that it is rare in hot countries ; and that it does not occur in Japan in spite of prolonged lactation. Maternal nursing and an open-air life in a mild climate have a greater prophylactic effect than mere locality.

Associated digestive disorders also indicate that the diet is unsuitable. If such disorder is severe or prolonged, the marasmic state hides the rachitic one. Marked evidence of the disease is rare in marasmic infants. This probably means that assimilation and growth must take place in order to produce the characteristic physical signs. Syphilis has no influence beyond interfering with digestion and assimilation, and reducing the resisting power of the infant. Tuberculosis has a similar effect. The malformation of the thorax induced by rickets, and the catarrhal affections of the lungs to which a rachitic baby is liable, render it more susceptible to tuberculosis. Only wasting disorders can be regarded as antagonistic, and they are merely so because the child has not sufficient vitality left for the production of the characteristic proliferative processes in the cartilages and bones.

*Morbid Anatomy.*—Changes take place in the bones as the result of imperfect nutrition or chronic inflammation, or a combination of the two processes. There is increased vascularity of the medulla and periosteum, and increased cell proliferation, but ossification is imperfect in spite of the excessive preparation made for it. The bones become soft and flexible, and the epiphyses enlarged. At the epiphyseal line of the long bones there are found excessive vascularity of the cartilage and abnormal proliferation and irregularity of the cartilage cells, and of the osteoblasts in the deep layer of the periosteum. The process of ossification is imperfect, slow or arrested, and the normal absorption of the inner layers of the bone in the medullary cavity is increased. At the growing zone calcification is deficient and much osteoid tissue formed. The cartilage at the epiphyseal line is thickened, bluish-white, softer than normal, and almost gelatinous. On one side it blends with the cartilage of the epiphysis, and on the other has an irregular dentate border and contains scattered irregular calcified areas. This causes beading of the ribs and enlargement of the epiphyseal line at the ends of the bones. In more severe cases the whole of the epiphysis is involved, for the ossifying centres become softer, larger and unduly vascular. The shafts are abnormally flexible, because of deficient periosteal bone formation and increased medullary absorption. Normal bone contains 37 parts of organic and 63 of inorganic matter ; the worst rachitic bone, 79 organic and 21 inorganic, in consequence of the absorption of phosphate of lime. Kassowitz states that the deficiency of lime salts is 20-30 per cent. The shafts become rounded and abnormal curves form. These curves are simple exaggerations of normal curves, produced by the weight of the trunk and limbs. Abnormal curves are due to unnatural positions and abnormal muscular action. Premature union of the shaft



with the epiphysis may arrest growth. Fractures result from trivial causes on account of the fragility of the bones. They are usually of the green-stick variety, at the point of the greatest curvature of the bone, and most common in the radius, ulna, clavicle and ribs in the order named; much callus is thrown out.

The enlargement of the cartilages at their junction with the ribs produces the "*Beading*" or rickety "*Rosary*." It is first felt at the ends of the fifth and sixth ribs, and is most marked on the pleural surface. The so-called beading at the back of the thorax is due to partial fracture of the ribs at or near the angles. These beads are angular, rather than nodular, projections on the external surface; single or unilateral, asymmetrical, and variable in position. In front of the chest grooves are situated outside the beads, extending from above obliquely downwards and outwards, and are due to the atmospheric pressure on the ribs which are softer than the cartilages.

*Harrison's sulcus* is a transverse groove at the level of the upper limits of the liver, spleen and stomach, which prevent the lower parts of the chest wall falling in as much as the upper. The liver renders it more marked on the right than on the left side. The unyielding abdominal viscera cause eversion of the lower ribs. On the left side the ribs may be a little more convex on account of the underlying heart. The apex of the heart may be displaced outwards; and the left ventricle be more exposed and exhibit a white patch due to attrition against a bead. There is partial collapse of the lung under the deep antero-lateral furrows, with emphysema of the anterior portions. Bronchitis, broncho-pneumonia and patches of collapse are often present.

The abdomen is "pot-bellied" from flatulent distension due to intestinal catarrh, excess of carbohydrate food and muscular weakness. The liver is sometimes enlarged, and still more often appears so, for the deformity of the chest exposes more of its surface. The spleen and lymphatic glands may be larger than normal. The pelvis is more triangular than oval in shape, but this depends partly on the age at which the disease is active and the position assumed by the child. The bodies of the vertebræ are softer and more vascular than normal. Between them and the discs is a bluish-white proliferating zone of cartilage.

The cranium may show extensive craniotabes from delayed ossification or a patchy variety due to re-absorption of already formed bone. Ossification of the fontanelles is commonly delayed. In mild cases the edges only of the cranial bones show periosteal thickening; in more severe ones cranial bosses or hyperostoses are formed. These are soft, red, vascular masses of spongioid bone, as much as half-an-inch thick, and can be indented with the finger or cut with a knife, blood and serum being easily squeezed out. They are due to thickening of the bone, chiefly the outer table, about the centres of ossification. In the course of time they are absorbed or



organised into light porous bone, or form diffuse laminæ of dense ivory-like bone. The abnormal ossification makes the appearance of the skull suggestive of hydrocephalus.

*Pathology.*—The cardinal factor is increased vascularity of the growing portions of the bones, giving rise to excessive cell proliferation and irregularities of calcification and ossification. When it subsides the vessels diminish in size and normal bone is formed. The process may be regarded as analogous to that of a chronic inflammation. In support of this view it may be pointed out that artificially produced hyperæmia causes proliferation of cartilage cells, absorption of newly-formed bone, and deficiency of lime salts; and, further, that bones affected with chronic inflammation are deficient in lime salts.

Undoubtedly there is perversion of tissue metabolism. The deficiency of lime salts in the bones has been ascribed to deficiency of lime and phosphoric acid in the diet, and defective absorption or increased katabolism (*The Lime Theory*). Experiments on animals show that a diet deficient in lime produces osteoporosis and rachitic-like changes in the growing cartilages and the periosteum. This condition, however, differs from rickets, in that the soft tissues are also deficient in lime and that it can be cured by giving calcium chloride. It may also be urged against the lime theory that uncalcified cartilage is in excess in rickets, that such cartilage contains a high percentage of lime salts, and that marked proliferation of the cells is impossible without a supply of lime. Both cow's milk and farinaceous foods contain much lime.

According to the "*Lactic Acid Theory*," lime is absorbed from the bones by lactic acid circulating in the tissues, and there is an excess of lactic acid and other inorganic acids in the blood. These organic acids are produced by an excess of carbohydrate food and deficient oxidation. Consequently rickets develops in infants over-fed on carbohydrate food and deprived of sufficient exercise and fresh air. Deficient metabolism of carbohydrates leads to the production of by-products which affect cartilage and bone, as well as the general metabolism of the body. The administration of lactic acid to flesh eating animals produced rickets, and subsequently osteomalacia, and in herbivorous animals osteomalacia without antecedent rickets (Heitzmann). These experiments and conclusions are of doubtful value, for the food was deficient in lime salts and marasmus and other complications occurred. Korsakow repeated them on dogs without success. Wegner produced rickets by giving small doses of phosphorus, probably through its action as an irritant. We may conclude, therefore, that the various theories, viz., (1) errors of lime metabolism; (2) excess of lactic acid; (3) deficient blood alkalinity; (4) retention of carbonic acid, are all unconfirmed. Our knowledge is at present limited to the fact that the disease is the result of an improper diet, apparently one defective in fat, which gives rise to increased vascularity; and that both this and



the bone changes are due to the circulation in the blood of some irritant, which leads to absorption of lime and other changes. This is seen in the production of craniotabes by softening of previously ossified bone. If from deficiency of lime in the food, or from mal-assimilation of lime, the blood is defective in this salt, it is reasonable to assume that it re-absorbs lime from bone in order to maintain its normal equilibrium.

*Symptoms.*—The indications of rickets consist of certain constitutional symptoms and the anatomical effects, softening and deformity, on the skeletal system. The first signs are fretfulness, restlessness, disturbed sleep, throwing-off the bed-clothes, sweating of the head, beading of the ribs, craniotabes, constipation, and disinclination to move about if able to do so. Later on there develop enlargement of the epiphyses, especially the radial; deformities of the head, limbs and thorax; protuberant abdomen; weakness of the muscles, ligaments and other tissues of the body; anæmia; irritability of the nervous system; liability to catarrh of the respiratory and the alimentary mucous membranes; irregular and delayed dentition; and perhaps enlargement of the liver and spleen.

The blood shows nothing characteristic, though anæmia is often present and the hæmoglobin may amount to only 60 per cent. The anæmia must be regarded as due to adventitious causes, or as a result of the same cause that produces the rickets. It is not due to rickets itself, for ordinarily the number of red cells and percentage of hæmoglobin is increased. Nucleated red cells, polychromatophilia and myelocytes are rare; and the white cells show no definite change. Pyrexia, if present, is due to some complication. Some children are rosy and well nourished; others are pale, fat and flabby, with very little resisting power, and liable to acute affections of the mucous membranes. These are spoken of as cases of "*fat rickets*," as opposed to "*thin rickets*," in which there is considerable wasting. The appetite is generally good, sometimes it is excessive, and there is a great desire for salt. Constipation is frequent in the early stages and still more so in the later ones. It is due to lack of muscular tone and sometimes chronic intestinal catarrh; it may alternate with diarrhœa, for the hard dry stools set up a chronic catarrh in the colon, with passage of large quantities of mucus; the stools are often deficient in pigment. Diarrhœa is liable to be acute. The urine is faintly acid and deficient in calcium, but no marked differences have been noticed. Neither lactic acid nor phosphoric acid has been found in excess. Various symptoms arise from defective nutrition of the central nervous system. Sweating is profuse during sleep; the sweat is acid, stands out in drops on the forehead and may soak the pillow. Sometimes there is increased general sweating, with sudamina. Insomnia is common. While asleep the child is restless, rolls the head from side to side, rubs the hair off the occiput, burrows in the pillows, tosses about and throws off the clothes. These symptoms are not always due to rickets. There is increased susceptibility to reflex



irritation ; increased liability to muscular spasm in the form of laryngospasm, tetany and general convulsions. These affections are most common at 3-9 months of age, and often induced by stomach or intestinal disturbance. In some cases facial irritability is the only sign ; it must be regarded as a danger signal.

The head appears too large actually or in proportion to the rest of the body. Occasionally it is dolichocephalic, elongated fore and aft, with raised prematurely ossified sutures ; a type more common in the degenerate than the rachitic. Usually it is an irregular square with flattened crown and broad square forehead ; sometimes the frontal and parietal bosses are so marked as to stand up above the median and transverse grooves, due to the sagittal and coronal sutures, forming the “ *hot-cross bun* ” type, or “ *tête carrée*.” These bosses may have a pale bluish tinge, as seen through a thin scalp. They are a possible source, while growing, of local discomfort and irritability, leading to head banging, rolling, etc. The occipital protuberance is prominent, for the whole of the upper part of the bone may be flattened from pressure, so as to appear nearly vertical on side view. Marked asymmetry is due to flattening of the parieto-occipital region on the one side, and prominence of the frontal region on the other. The anterior fontanelle is large, and may remain open until the end of the first dentition, instead of being closed by the end of the eighteenth month. Even at the age of 2 years it may measure 2 ins. across, and there is a depression sometimes in its place up to the age of 5 years. The other fontanelles and sutures remain open an unduly long time, and there is delay in ossification of the bones along the edges of the sutures, giving rise to one variety of craniotabes. The superficial veins of the scalp are prominent and easily seen through the thin skin. They may form definite grooves in the bone, distinguishable with difficulty from irregular sutures. The hair is thin and often worn off the occiput, and sometimes off the sides, from sweating and friction.

The face looks small by contrast with the prominent forehead and large head. The antero-posterior diameter of the upper jaw is lengthened, and gives it a beak-like aspect ; that of the lower jaw is shortened, and the shape is irregular.

*Craniotabes*.—There are two distinct forms of this condition. The first is uncommon ; is seen in infants under 6 months of age ; and consists of small soft spots, round or oval,  $\frac{1}{4}$ -1 in. in diameter, and chiefly in the posterior half of the parietals and the membranous half of the occipital bone. On pressure they yield with parchment-like crackling. This variety is seen in congenital syphilis, with or without rickets. In the other type the free margins of the flat bones of the skull, especially the parietal and the occipital, are soft and late in ossifying, and remain membranous for a long time. It is quite common to find softening along the margins of the parieto-occipital suture, and in severe cases along the margins of



each cranial suture, and round each fontanelle. In the latter instances there may be isolated soft spots, due to surrounding ossification. The occiput is flattened. The condition is rather one of marked flexibility than true softening. This flexibility is found from the third to the twelfth month of life, rarely earlier and never later. It is most common in infants brought up on condensed milk, and in those seen in the early months of the year. It is due to delayed ossification or to re-absorption of lime salts from the newly ossified bone, in consequence of deficiency of lime salts in the food or their presence in an unsuitable form. It may exist without any other evidence of rickets, or with very slight beading. Consequently, it cannot be regarded as a definite proof of rickets, but it shows that the diet is unsuitable. On the other hand, it is cured by anti-rachitic remedies and is often associated with laryngospasm. The cranial bones may be membranous at birth and remain so, or undergo softening from cerebral tumour or hydrocephalus.

Variations in *teething* depend on the date of origin of the disease. The first teeth are often cut at the usual time, and the subsequent ones are delayed, cut irregularly, or "cut on the cross," or follow each other in rapid succession. Delayed dentition is by no means a proof of rickets, for infants of 12 months without teeth may show no signs or symptoms of the disease. On account of malformation of the jaws and impaired alveolar development, the crowns of the teeth are not properly adjusted and the incisors often bend inwards. The teeth seem especially liable to decay and discoloration, and often show horizontal ridges and depressions on the enamel. It is uncertain that this dental state is of rachitic origin. The milk teeth ought not to suffer, for calcification is advanced at birth, yet early decay of these teeth is common. The permanent teeth calcify during the first year of life and are more likely to be affected. Teething is more likely to cause illness than in normal children, because of the nervous instability and liability to catarrh of the mucous membranes.

The *beading* or *rachitic rosary* is the first and most constant anatomical change. The beads or nodules are most obvious on the fifth or sixth ribs, easily felt if not visible to the eye, and vary in size. They are rarely felt before the third month, sometimes at 1 month, and occasionally are present in the newborn and on the pleural surface in stillborn infants. Slight projections at birth must not be regarded as rachitic. In untreated cases the beads increase in size up to the end of the second year and then slowly disappear. Except in recrudescent or late rickets, there is little trace of them after the age of 4 or 5 years, and none at all in adults. A spurious beading on the pleural surface is due to partial dislocation of the ribs backward, at the costo-chondral junction. Posterior beads on the back of the thorax are due to green-stick fracture of the ribs near the posterior angles.

The chest appears very small in its upper part, in comparison with the



large square head and protuberant belly. The shape is altered by the action of the atmospheric pressure on the yielding chest wall at the points of least resistance, i.e., just external to the beads, causing a lateral flattening or broad shallow concavity. Deformity is increased by respiratory obstruction, giving rise to pigeon-breast, a peculiarity not found in uncomplicated rickets. The pressure of the arms and the dorsal decubitus also modify the shape of the softened yielding bones. The ordinary rachitic chest is fiddle-shaped in transverse section, increased antero-posteriorly and narrowed transversely by the sinking in of the lateral furrows. The sternum and cartilages are convex forward. In a few cases the sternum is depressed between the projecting cartilages. A slight depression may be seen or felt anterior to the rosary. Extending across the lower part of the thorax transversely is a groove or depression, called the "*rachitic girdle*," or "*Harrison's sulcus*"; it is most marked on the right side and deeper on inspiration. Breathing is increased in frequency, and chiefly diaphragmatic if the thorax is much affected, and the area of cardiac dulness may be enlarged.

The spine shows no change in mild cases. In severe ones it projects as a gradual kyphotic curve in the dorso-lumbar region, from the middle of the back to the sacrum, when the child sits up. The curve is due to laxity of the ligaments and generally, but not invariably, disappears if the child is suspended or laid on its belly with the legs extended. If it exists in a severe degree for a long time it may cause permanent deformity. This type of rachitic curve is not infrequently mistaken for commencing spinal caries. Lateral curvature, commonly convex to the right in the dorsal region, in children under three is usually rachitic, and occasionally associated with some rotation of the vertebræ.

The anterior diameter of the pelvis is often diminished, and the suprapubic arch narrowed. The iliac crests are thickened, and occasionally the pelvis is very irregular in shape or "*crumpled*." These changes are due to the weight of the body and abdominal viscera, and lead to subsequent difficulties in labour.

The clavicle may be thickened at the sternal end, prominent about the middle from green-stick fracture, or project upwards and forwards at the junction of the middle with the inner third, due to muscular traction and weight of the arm. The infra-spinous fossa may be unduly convex. In an infant, who crawls on the hands and knees or supports itself on its hands, the humerus develops a forward and outward curve about the attachment of the deltoid. The epiphyses are enlarged, the lower one being the more easily felt. Much epiphyseal change takes place in the radius and ulna, especially at the lower end, and in crawlers these bones develop marked curves and are very liable to green-stick fracture. Pronation of the hand results from a spiral bend in the radius. The hands are not characteristic. The fingers are somewhat hyperextended and may seem beaded, from the



joints being smaller than the middle of the phalanges. Deformities of the metacarpals and phalanges are rare, but the latter may appear thickened and spindle-shaped. Neurath states that periosteal thickening can be demonstrated by X-rays. I doubt there being any actual thickening.

In the lower extremity deformity is more common, because of the weight of the body in standing and walking. The femur develops a forward and outward curve. If the child is allowed to sit cross-legged, the bone is rotated outwards and the toes are much turned out in walking. Coxa vara is due to the weight of the trunk. Knock-knee is more common in females, and is either due to overgrowth of the internal condyle or weakness of the lateral ligaments. Bow-legs in slight cases are apparent, not real, being due to enlargement of the epiphyses. In children who stand or walk, there is actual curvature of the bone outwards, and this is often associated with an antero-posterior curve, usually situated and perhaps extreme at the junction of the middle and lower thirds of the bones. Bowing is not always equal in the two legs. Sometimes it is unilateral and associated with knock-knee on the other side. In rare instances it is due to subperiosteal fracture. Even in severe cases the bones of the foot are not appreciably affected. In the lower limb the enlargement of the distal end of the tibia may be the only indication of rickets.

It is clear that slight deformities of the extremities are due to epiphyseal overgrowth, and that the more severe and more permanent ones are produced by bending of the shafts under the superincumbent weight, due to crawling, standing, sitting and walking. Growth may be arrested, especially in the lower extremities.

Laxity and elongation of the ligaments produce weak ankles, flat-foot, over-extension of the knee (calf knee, genu recurvatum), knock-knee, spinal deformities, and unnatural mobility of joints or *acrobatic rickets*. Possibly this type is entirely due to hypotonicity of muscles. The muscles are small, flabby, atonic and badly developed. The motor functions are impaired, delayed or lost. Hence arises delay in the child learning to sit, stand and walk. This is an advantage by preventing deformity. Late walking is common, and the child may not even attempt to walk until 3 years of age. Sometimes the muscles of the legs are so weak and atrophied that the child comes under treatment for supposed paralysis. The weakness may be very marked, though the skeletal changes are slight. The electrical reactions are normal or exaggerated.

The muscles of the abdominal wall and of the alimentary canal are weak; the contracted chest pushes down the abdominal organs; and the pelvis is flat. Flatulent distension is common because of disordered digestion. Hence arises a condition of "*pot-belly*," or "*frog-belly*," in which the abdomen is uniformly enlarged, tense and tympanitic, the recti often separated (diastasis of the recti), and an umbilical hernia may be present.



The stomach and colon are much distended from fermentation of their contents.

Apparent enlargement of the liver, which is normally large in infants and projects into the abdominal cavity because of the more horizontal position of the lower ribs, is due to the rachitic deformity of the chest and laxity of the ligaments, downward displacement and greater exposure of its surface. True enlargement may occur, and the liver is smooth and firmer than normal. It is due to a portal cirrhosis, interlobular in type, set up by toxæmia resulting from the gastro-intestinal catarrh so often present. Hogben (1888) described a hypertrophic or biliary type, monolobular and interlobular, with an increase in the number of small bile ducts. It is difficult to accept this explanation for jaundice does not occur.

The spleen can often be felt, for it is displaced downwards and more exposed by eversion of the lower ribs. Possibly a simple hyperplasia occurs during the acute stages, and fibroid changes later on, as the result of associated toxæmia conditions.

*Course and Prognosis.*—The disease is very chronic and lasts for months, or even more than 2 years. Its course is modified by treatment. Sometimes it is latent, or there may be exacerbations. Usually the active symptoms cease when a mixed diet is given, that is about the end of the first year. Different parts of the skeletal system are progressively involved, recovery taking place in one while another becomes affected. Thus, in the first 6 months craniotabes and beads may be the only signs; later on the thorax is affected; then kyphosis, bony deformities, etc., arise. Signs of improvement are diminished sweating and restlessness, disappearance of craniotabes, increase in muscular power and improved general nutrition. Deformities slowly but steadily improve, except in the worst cases, in which they may persist throughout life in the modified form of pigeon-breast, Harrison's sulcus, eversion of the costal arch, kyphosis, pelvic deformities, and knock-knees or bow-legs. Relapses are rare.

It is never fatal, *per se*, but is the cause of increased mortality from other diseases, especially those of the respiratory and alimentary system. Laryngitis, bronchitis and broncho-pneumonia are much more fatal in the rachitic, because of the weakness of the respiratory muscles and the thoracic deformity. Death may also result from laryngospasm, convulsions, tetany or gastro-enteric complications. The deformity of the chest retards growth by interfering with efficient lung expansion and oxidation. Apart from this deformity the disease has no permanent influence on the general health. A child may be stunted from arrested growth at the epiphyses. The mental development may be retarded by malnutrition or on account of the physical disabilities.

*Diagnosis.*—Beading of the ribs and enlarged epiphyses are diagnostic. Restlessness, head sweating, irregular dentition, craniotabes, a large fontanelle and backward development may be due to other causes, but in



conjunction are strongly suggestive. A large fontanelle and delayed dentition may be due to cretinism ; a large head, to hydrocephalus ; delayed walking, to backwardness ; and muscular weakness, to paralysis. Curvature of the spine may simulate caries. If the bony changes are slight and the muscular weakness great, it is difficult to be certain that all are due to rickets ; even if the bony changes are well marked, the extreme weakness may suggest an additional affection. The presence of knee-jerks and normal electrical reactions distinguish the muscular weakness from that of infantile palsy ; while exaggerated reflexes and rigidity indicate cerebral paralysis. The large belly may simulate tabes mesenterica. Achondroplasia causes stunting and false beading. Syphilitic epiphysitis is generally monarticular and in infants under 6 months of age. The pain and tenderness of scurvy must not be ascribed to rickets. The rachitic element may lead to errors in diagnosis, if present in other diseases. Many nervous and catarrhal conditions have their origin in this disease. Mentally deficient children are often rachitic, but the backwardness must not be ascribed to the rickets.

*Recrudescent Rickets* sometimes called "*Adolescent*" or "*Late*" *Rickets*, is rare. Charles West stated he had never seen the disease begin later than 3 years of age. Sir William Jenner referred to a case in a girl, aged 9 ; and since then at least 30 cases, some of them doubtful, have been reported. Two-thirds of the cases are girls. In a girl, aged 11 years, who came under my care in 1896, the disease was said to have begun at the age of 4 years ; she was anæmic, wasted, extremely feeble, and had irregular fever, up to 102-103° F. for 16 days. Under treatment there was very great diminution in the size of the beads and epiphyses. The disease appeared never to have subsided since its onset, but to have remained quiescent. We can divide the cases into three types. In the first there is a recrudescence or relapse, the disease never having been completely in abeyance ; in the second type there has been a true recurrence ; and in the third the patients are said to have been unaffected in infancy. The common age of onset is 12-18, and in a few at 6-11 years of age. Ossification is not complete at this time and rapid growth takes place at puberty. One of Clutton's cases showed premature sexual development. The affection comes on with stiffness and pain in the joints and leg muscles, and the gait may suggest hip disease. This is followed by enlargement of the epiphyses and beading. The skull is unaffected. The deformities include flat-foot, genu valgum and varum, various femoral and tibial curves, coxa vara and scoliosis. In a case of Little's (1900), a boy, aged 17, with symptoms since the age of 5, the epiphyses were swollen and the bones soft, showing irregular ossification and islets of ossification typical of rickets. It may be more or less unilateral, but the diagnosis in cases of limited distribution must be accepted with caution. Possibly some of the recorded cases are instances of osteomalacia, from which it can be distinguished by the disturbance of endochondral ossification.



*Intra-uterine Rickets*, sometimes called "*Fœtal*" or "*Congenital*."—It is doubtful whether true intra-uterine rickets has ever occurred. An open fontanelle, soft bones, costo-chondral swellings and protuberant belly occur in all infants. The results of histological examination of the bones and cartilages are not accepted by some observers as characteristic of rickets. Charrin and Le Play (1905) ascribed the rosary, craniotabes, enlarged epiphyses, etc., in a child born at 8 months, of a mother aged 47, who had been subject to privations, to true rickets. They based their opinion on macroscopic and microscopic examination and chemical analysis. It has been divided into two classes, the *fœtal* form, which develops and is cured during intra-uterine life, and a *congenital* form. Possibly it may be present and give rise to no definite signs until sometime after birth. It must not be confounded with achondroplasia, nor with syphilitic osteochondritis, and it is certainly not justifiable to accept beading of the ribs in the newborn as proof of rickets. Fractures in utero are commonly due to osteogenesis imperfecta.

*Treatment*.—Prophylaxis consists in care of the mother during pregnancy and lactation, suitable diet for the child, fresh air, sunlight, a daily bath, and exercise or massage. The value of drugs is difficult to estimate, for active symptoms usually cease at 12-18 months of age, and even if untreated the disease subsides when the child obtains a mixed diet. Mild cases recover without any special treatment. If the child is not breast-fed, the diet must contain a liberal proportion of fat and protein in the form of cream, milk, eggs, red meat, butter, bacon fat, and suet, if suitable at the age of the child. It is generally advisable to diminish the amount of carbohydrates and allow a liberal supply of salt. There is probably enough phosphoric acid in the tissues to seize on any spare sodium.

Let the child have light and air in the nursery, and be out in the open air and sun as much as possible. Country air is better than urban, but there is no special virtue in the seaside. A daily salt bath (a handful of rock salt to a bucketful of water) at a temperature of 70-80° F., followed by dry rubbing, is a useful stimulant and diminishes the liability to taking cold. On account of the susceptibility to chills, the neck, legs and arms should be covered, and at night the child should wear a flannel nightdress, tied below the feet, or pyjamas and socks. The pillow must be firm and not soft. If the alimentary track is out of order and fermentation has induced atony, flatulent distension and separation of the recti, the diet must be reduced and a mixture given of ol. ricini m. 10-15, tinct. rhei m. 5-10, tinct. nucis vomicæ m.  $\frac{1}{2}$ -1, and sod. sulphocarb. gr. 3-5, t.d.s.

Cod-liver oil is the best drug, alone or in combination with maltine and syrup ferri phosph. co. Phosphate, lacto-phosphate and hypophosphites of lime do not appear to have much value. A suitable mixture is that of calcium and sodium hypophos. aa. gr.  $\frac{1}{2}$ , ol. morrhuæ and glycerin aa. m. 20, ol. cassiæ m.  $\frac{1}{10}$ , and tragacanth mucilage to 1 dr. Phosphorus



has been called by Kassowitz "the iron of rickety children." It is given in daily doses of  $\frac{1}{2}$ -1 dr. of phosphorus gr.  $\frac{1}{8}$  in 3 oz. of cod-liver oil, to which the credit should be given. Suprarenal extract is also said to exert a favourable influence on the craniotabes, sweating, nervous manifestations, and general condition of the child. Arsenic and iron are useful if there is much anæmia.

Respiratory exercises assist in the proper expansion of the chest, but are distinctly injurious if it is found that the chest wall sinks in on inspiration. A firm abdominal bandage is of some assistance. Adenoids and enlarged tonsils must be removed. For lordosis the child must sleep on a hard level bed, with a pillow under the spine if the curve is severe. For a few minutes daily it should be placed on the belly with buttocks elevated, while pressure is applied to the spine. Massage of the spinal muscles is useful, and a spinal support is permissible, to allow the child to sit up for short periods at a time. Sitting must be prohibited if the disease is very active, for fear of pelvic deformity. Bowing of the legs is best treated by massage. The best splints are made of poroplastic felt. They are made to extend from a little above the knee to the ground, and fastened by a strap round the ankle, another below the knee, and a third round the middle of the calf. The last one is of chief importance. It is fastened round a square leather pad on the outer side of the calf and tightened daily. These splints are easily put on and off, can be kept clean, and do not interfere with exercise. External splints are permissible, if they extend 2 or 3 ins. below the feet and are of uneven length, and thus keep the child off its feet. They must be worn at night, if the limbs are curled up in bad positions. This treatment may increase the pressure on the upper limbs, for the child crawls about on the hands and knees. It is most suitable when the disease is stationary in the upper limbs though progressive in the lower ones. Slight bowing, which does not increase, requires no special treatment. A tracing of the limbs on paper, when the child is first seen, enables one to estimate any increase in curvature on a subsequent visit. It is sometimes recommended that deformities should be treated by osteotomy after the age of 3 years, on the ground that delay only perpetuates the condition and the changes in the bony surfaces and ligaments. It is better to delay operative treatment until the seventh year, for many bad cases get well, as the result of the natural tendency of bone to assume the normal shape. Knock-knee in the early stages is treated by massage and an external splint, with a certain amount of pressure exerted by a bandage over the knee. In late stages operation is necessary.

The essential treatment of the disease is preventive. If it has once commenced proper diet and management prevent disastrous results. A marked case of rickets in private practice is a grave reproach to the regular medical attendant.



## CHAPTER XVI.

### DISORDERS OF BONE FORMATION.

*Achondroplasia—Cleido-Cranial Dysostosis vel Anosteoplasia—Osteogenesis Imperfecta vel Periosteal Aplasia.*

The nomenclature of these various affections has caused considerable confusion, for it is by no means uncommon to find different diseases described under the same name.

It would be advisable to make use of the term "Osteogenesis Imperfecta" as a generic name for the group of bone affections in which ossification is defective. To simplify the nomenclature we might speak of *Achondroplasia*, an affection in which those bones developed in cartilage are chiefly at fault; *Anosteoplasia*, a condition the antithesis of achondroplasia, in that the defect is in the development of the membrane bones; and use the name, *Periosteal Aplasia*, for those cases at present described under the title of Osteogenesis Imperfecta, for in this the developmental defect is apparently in the periosteal formation of bone, that is, in the osteogenetic layers of the periosteum. I have adopted this nomenclature in the titles of these diseases.

**Achondroplasia.** — *Syn: Chondrodystrophia Fœtalis—Congenital Rickets.*—Parrot, in 1878, gave the name of achondroplasia to a foetal systemic bone disease, with characteristic clinical features, definite pathological anatomy, and of unknown causation. Since Kaufmann's monograph (1892) it has been known as chondrodystrophy foetalis, but it has frequently been described under other names. It affects the cartilage, but not the membrane bones, and has nothing in common with rickets or cretinism.

Although the first case was recorded by Sömmerring, in 1791, it is a disease of great antiquity and is represented by the Egyptian gods, Pthah and Bes; by dwarfs, in pictures by Velasquez; by some statues of Silenus; and by the effigy attached to the tomb of Knoumhotpon, chief cook of Pharaoh, in the necropolis of the School of Memphis. The disease is not uncommon and probably occurs in calves, sheep, goats and pigs; but some of these so-called cases in animals are monstrosities. Frequently it is associated with hydramnios, abnormal presentations, and prematurity. Or the infants are stillborn, or only survive a short time. Their hold on life is precarious at first. Later on they are as strong as ordinary children.



The characteristic feature is stunting of the upper and lower limbs, the vertebral column being unaffected or even abnormally long. At birth the head is large, and appears still more so on account of the shortness of the limbs. Its circumference equals or even exceeds the length of the body. It is brachycephalic, globular, with projecting forehead and flattened nasal bones. The arms do not reach the waist, and the shortness of the lower limbs causes the navel to be lower than the mid-point between crown and sole, its position in normal infants. The mid-point may be as high as the xiphoid cartilage. The limbs are often bent, deformed, and occasionally fractured. The epiphyses are normal or appear enlarged, and the costochondral articulations are thickened, in "rosary" style. As a whole the infant is obese, with prominent belly, thickened skin and subcutaneous tissues, and fine, soft, abundant hair. The eyelids are thick and there may be epicanthus. The lips and tongue are thick, and the tongue protrudes. The nose is retracted, and there may be a deep depression at its root or marked flattening of the whole nasal region, with decided prognathus. The face is relatively small. Thus, there is a close resemblance to the cretinoid state, and less so to rickets.

Survivors are apt to be mistaken for cretins; but can easily be distinguished therefrom. They are quite as intelligent as other children of the same age, except that they may be a little backward through being kept away from school. The disease is over, though its effects remain. They may be described as obese, micromelic dwarfs, with broad heads, long bodies and short limbs, who attain a height of 3-4 ft.

They are often bow-legged, and their gait waddling or quite natural. The fontanelle is large, and remains so for a long time; the palate rather unduly arched, but dentition is normal. Hydrocephalus is not uncommon. The shortening especially affects the femora and humeri. The pelvis is small and contracted, with the sacrum projecting strongly forward and the spine in a position of marked lordosis. The fingers are stunted, the three middle ones of equal length, divergent, curved, and cannot be easily approximated; the toes are short. The child may be quite robust and able to walk at an early age, but more often walking is delayed. Obesity disappears as it gets older. Inguinal hernia is frequent.

Instances of heredity have occurred. Seligmann has pointed out that achondroplastic Dexter-Kerry cows may have cretinous calves. Shattock regards it as a para-cretinous condition; but the thyroid gland is normal. It has been ascribed to inflammation of the cartilages or an anomalous form of osteomyelitis in the first months of foetal life. The vascularity of the growing cartilage is always increased, but bone is imperfectly formed and ossification arrested. There is a deficiency or absence of the proliferating zone of cells at the epiphyseal line, and more or less complete inhibition of the normal arrangement of the cells in columns preparatory to ossification. Bone formation is periosteal; and the bones become



very dense, compact and bossed. The beads on the ribs are due to a bony investing ring derived from the periosteum. The cartilage bones at the base of the skull are stunted in consequence of premature synostosis of the basi-sphenoid, basi-occipital and præspenoid. These bones form the *fundamental bone* of Hyrtl or the *os tribasilar* of Virchow, which may be shortened independently of premature union, and is not shortened in every case. To this shortening are ascribed the prognathus and depression of the nasal bridge. The membranous bones of the skull and the clavicles, ossified very early in foetal life, are unaffected.

Kaufmann has described three varieties, all of which may be found in the same individual, though one usually predominates. Rudimentary row formation is present in all. In *Chondrodystrophia Fœtalis hypoplastica* the epiphyseal cartilages are diminished in consequence of deficient proliferation of cartilage cells. Miss Mowler, in Dicken's book, "David Copperfield," is a case of this type. In *C.F. hyperplastica*, there is great proliferation of the cells and enlargement of the epiphyses. In *C.F. malica* the cartilages are much softened, and the epiphyses more or less gelatinous. Each variety shows increased vascularity in the cartilage. Periosteal bone formation and the deposition of calcium salts go on normally. A periosteal lamella or inclusion has been described in the human foetus (Urtel), and in the calf (Eberth). This is a layer of connective tissue, starting from the periosteum, making its way in between the epiphysis and diaphysis, and more or less separating them. Its extent varies from a mere trace to a definite partition reaching to the axis of the bone. By encroaching upon the epiphyseal line, it precludes growth in length and may entirely stop the growth of the bone. It is the main cause of the physical peculiarities.

It is of the utmost importance to differentiate this affection from cretinism in early life. The main differences are an unimpaired mental state, a normal thyroid, a periosteal lamella, and the absence of myxœdema. Thyroid treatment is of no benefit. The bones of cretins show nothing abnormal beyond exceedingly slow ossification.

**Anosteoplasia.** — *Syn. : Osteodystrophy Fœtalis — Cleido - Cranial Dysostosis.*—Seeing that this affection is the antithesis of achondroplasia or chondrodystrophy foetalis, it may be named anosteoplasia or osteodystrophy foetalis, rather than cleido-cranial dysostosis under which it is generally described. Scheuthauer (1871) noted the connection between the deformity of the cranium and the clavicles, but the first full description was given by Marie and Sainton, in 1897. There are now about 40 cases on record.

The disease is hereditary, and may affect father and son, or mother and daughter ; or may occur as an isolated peculiarity. It is a defect in the development of the membrane bones. The ossification of the membrane bones of the skull is much retarded, and the fontanelles may remain open



till a late age. The skull is globular, brachycephalic, with frontal or parietal bosses. The face, nasal bones, jaws and mastoids are small; the palate high and deficient; the teeth few, irregular and delayed; the clavicles rudimentary, atrophied or absent.

The clavicle is said to be developed in membrane (Bruch), in cartilage (Kolliker), or mixed (Gegenbauer). That it is developed in two parts is shown by cases in which the outer half is absent; others in which there is a rudimentary inner or outer part, or two contiguous but unjoined halves, forming a kind of loose joint. The bone may be completely absent; form short, floating osseous bands, partly rudimentary and attached by a fibrous band to the coracoid; or almost absent and contain no bone. The clavicular defect enables the shoulders to be approximated in front.

The dystrophy is not limited to the membrane bones. It may affect the general growth and be associated with dwarfism and infantilism. Intellectually and psychically these children are normal.

**Periosteal Aplasia.**—*Syn. : Osteogenesis Imperfecta—Fragilitas Ossium—Infantile Osteomalacia—Brittle Bones—Osteopsathyrosis.*—This affection is a systemic bone disease, which commences in early foetal life and is usually fatal before birth. It is characterised by multiple fractures and extensive craniotabes, due to defective ossification of the diaphyses and of the bones of the cranial vault. The epiphyses and basis cranii are normal.

The first case was described by Berdenave in 1763, and the name given by Vrolik in 1849. Up to 1906 about 130 cases of multiple spontaneous fractures, probably due to this cause, had been recorded. Most of the cases are those of premature or stillborn children, and it has been generally assumed that the affection is incompatible with extra-uterine life. In some instances the fractures did not begin until a few months (3-9) after birth. Other cases of fragilitas occur later in life, coming on about puberty, and rarely earlier, and are probably of different pathology. Fractures may also occur in scurvy and rickets in consequence of bone atrophy. A true mollities ossium or osteomalacia has also been reported as early as the fifth year of life; and a rachitic softening, due to the formation of spongy bone containing little or no lime salts, in the "limeless bone" variety of rickets.

Nothing definite is known about the etiology, except that it is not due to congenital syphilis, rickets or nervous lesions. Heredity has been noted in 15 per cent. (Griffith, 1897). It has been transmitted through three generations (Harmer); and it has affected 9 out of 10 children, and a child of one of these.

The bones are delicate and fragile, and broken with extreme ease. Sometimes the periosteal shell can be crushed between the finger and thumb. On section they are found very porous, with thin defective



trabeculæ, and a thin outer periosteal layer of small imperfectly laminated bone plates or trabeculæ, and large marrow spaces instead of Haversian canals. The marrow is composed of œdematous myxomatous connective tissue. Calcification is defective, and new bone is formed by direct conversion of cartilage cells, and not by the deposition of bone by osteoblasts on a previous cartilaginous matrix. The disease is confined to the bones of the cranial vault and the shafts of the bones. It prevents normal development as soon as the stage of true bone formation is reached. Apparently it is a defect in periosteal bone formation.

The newborn infant is usually very weak and, if handled with great care, may escape fracture for some days or months. More often fractures are present at birth. In appearance the child may be normal, smaller than normal, or look obese and œdematous from thickening of the skin and subcutaneous tissues. In this respect it is rather like achondroplasia, but the extremities are not stunted, except as a result of fractures. The limbs may be bent and deformed, and the bones so brittle that they fracture on the least provocation, or unduly soft and bend without breaking. As many as 113 fractures have been counted in a newborn child (Chaussier). They may be so numerous that the callus thrown out makes the bone nodular. In the ribs this may give rise to spurious beading. False joints are rare. In order of frequency the thigh, leg, upper extremity, clavicles and ribs are affected; rarely the hand and lower jaw. They unite readily with formation of much callus. The head is large, sutures widely open from deficient ossification, and exhibits craniotabes or is even completely membranous. The fractures are complete and rarely preceded by pain, though it has been recorded, and there is little pain at the time or during the process of healing; no doubt on account of the slight shock and damage to the soft parts. Marked deformity results from malposition and bending, perhaps inability to walk or stand, and scoliosis. After the first few months of life the general health is unimpaired, and the intelligence normal; nearly all learn to walk. Irregular fever and pain on movement, before fracture occurs, have been reported in some cases. X-ray examination:—the medullary cavity appears proportionately large; the epiphyseal line sharply defined; and the shadow of the shaft feeble or unduly translucent.

The disease persists throughout life and affects the whole skeleton. Probably the active process subsides during intra-uterine life or soon after, and the fragility is simply the result. Cases vary in degree, some are mild, while in others the liability to fracture is so great that the patient is confined permanently to bed; crippling is common. Many die from complications within two years of birth, and others have lived to 30 years. In those that survive the skull bones become ossified and the liability to fracture gradually becomes less. The diagnosis from achondroplasia is simple. Craniotabes with false beading of the ribs may be mistaken for



rickets, but in this the epiphyses are enlarged. Osteomalacia begins about or after puberty. The liability to fractures in congenital syphilis also begins about puberty, and more violence is necessary. Nothing can be done for these patients except placing them in a position to avoid fracture, protecting the limbs, and attending to the general health.



## CHAPTER XVII.

### ANOMALIES AND DISORDERS OF GROWTH.

*Dwarfs — Symptomatic Infantilism — Ateleiosis — Precocity — Obesity — Gigantism — Hemihypertrophy.*

A normal or physiological dwarf is an undergrown child or adult, not deformed, whose development has proceeded symmetrically at a normal rate, except as regards extent in comparison with other races, families of the same race, or members of the same family. The best illustration is the race of pygmies in Central Africa, whose height is about 4 feet. It is not due to any pathological process. Ossification, epiphyseal union and sexual development take place at the usual time.

A phocomelic dwarf is one in whom intermediate parts of the limbs are absent. In micromelic dwarfs the limbs are unduly small, e.g., achondroplasia. True dwarfs or Nanasomes are of two types: (1) *Primordial* or *sexual dwarfs*, in other words, small adults; and (2) *Infantile* or *asexual dwarfs*, i.e., permanent children. To the group of nanasomes Hastings Gilford has given the name of Ateleiosis, a Greek word meaning "not arriving at perfection."

*Symptomatic Infantilism* or *Dwarfism* is a term used for dwarfism associated with delayed ossification, dentition and sexual development. It is the result of some illness or general nutritional disorder, which interferes with growth. In these cases the development of the body is delayed or arrested. It remains small, weak and slender. Usually there is merely mental and physical delay, a variation within the limits of health. The common causes are general dystrophies, congenital heart disease, mitral stenosis, tuberculosis and congenital syphilis. It may be due to cirrhosis of the liver, "*Hepatic Infantilism*"; absence or deficiency of the secretion of the pancreas, the "*Pancreatic Infantilism*" of Byrom Bramwell; to malaria and poisons, e.g., alcohol, nicotine and lead; to rickets, microcephalus, spinal caries or lateral curvature; to thyroid insufficiency and to idiocy. Herter (1908) has described a "*Toxæmic Infantilism*" of intestinal origin. The abdomen is distended, and the stools contain much fat, soap, fatty acids and mucus. It is apparently identical with Bramwell's type, but Herter ascribes it to the prevalence of *B. bifidus* and *B. infantilis*, which replace the normal intestinal flora. In microcephalus the body remains small through an attempt to adapt growth to the size of the brain.



In congenital syphilis sexual development is commonly, but not always, arrested for a time. These patients ultimately reach maturity. Mongols generally remain small.

Thyroid insufficiency produces various types, of which cretinism is the most common. Its effect may be limited to a simple delay in growth or a general infantilism. Thyroidectomy in young animals causes dwarfism, sexual atrophy and deficient intelligence. Brissaud described an acquired myxædematous type, sometimes called *Myxædematous Infantilism*, in which physical development is arrested, the morphological characters of childhood persist after puberty, and general mental deficiency is often present. Another type, described by Loraine as due to congenital non-development of the arteries, is sometimes combined with thyroid insufficiency. Congenital diminution of the lumen of the larger arteries (Anangioplasia) has been found combined with lymphatism in the idiopathic epilepsy of Oldmacher.

*Lymphatic Infantilism* is a variety described in children of large French cities, and probably occurs elsewhere. The child develops rapidly and is precocious until the tenth or eleventh year, and then stops. He becomes lethargic and ceases to grow. The development of the testes is arrested, and the mammæ enlarge and sometimes form abscesses. He gets pale and fat, and is like a bag of fat. A violent shock to the circulation, such as a cold bath or douche, may cause sudden death. In this respect it is comparable with the status lymphaticus.

**Ateleiosis** is divided by Gilford into two groups : Asexual and Sexual. They are well-proportioned dwarfs with childish faces and intelligence, irregular and backward teeth, small bones and muscles, and an imperfect sexual system. It is of unknown causation. In the asexual type the whole body is affected, but the sexual organs are the most backward. It is not hereditary and is usually only seen in one of a family. It may commence before birth, in infancy, or at any stage of development. Hence its characteristics vary with the time of onset. Those affected are not necessarily dwarfs, since it may not begin until an average height has been obtained. The affection is one of delay rather than of arrest, for growth and development may proceed very slowly for many years after these processes normally cease, e.g. up to 30 years. The degree of delay is often irregular, some parts being more backward than others. In a case of Gilford's the actual age of the boy was 12 years, but the generative organs were like those of an infant at birth, the head and face like those of a 2-year old child, the bodily development that of a 3-year old child, the epiphyseal development that of 6 years, and the teeth only 1 year delayed. The thyroid seemed normal and there were no signs of cretinism. The general appearance of the patient is that of a much younger child. The body proportions, contour and intelligence are those of a child, and the testes are commonly undescended.



In sexual ateleiosis development is delayed until puberty. The epiphyses then unite and the sexual organs mature normally. The child resembles a miniature adult, but retains the physiognomy, proportions and stature of a child. Occasionally facial hair grows in the male. Although epiphyseal union takes place as usual, the bones do not grow in length and thickness at puberty. The muscular system develops well. These patients differ from normal dwarfs in the retention of many childish characteristics. They tend to die out. The affection is often hereditary and may affect more than one child in a family. Some of them have children of normal size, and occasionally have a child of the asexual type. Such an occurrence suggests that there is a close connection between the two varieties. Many of the notable dwarfs in history belong to one or other of these types of ateleiosis, while others are achondroplastic dwarfs. Some are said to have lived to extreme old age. Gibson, painter to Charles I, lived 78 years; Tom Thumb, 46 years; Boruwlaski, a Pole, died at Durham in 1837 aged 98; and Richebourg, a little over 2 feet in height, died in Paris in 1858 aged 90. Sexual immaturity delays growth and sexual hypoplasia precedes the skeletal defects, but ateleiosis must not be regarded merely as sexual infantilism. In this there is no special type of face, mental development is normal, and the sex organs are by no means infantile though delayed in development. Premature senile changes occur in both types of ateleiosis.

*Physical and Sexual Precocity* develop under various conditions and take different forms, viz.—

Premature sexual development.

Precocious obesity, with or without premature sexual development; in both sexes.

Abnormal strength and muscularity; in males only.

Hirsuties; present in almost all cases and not necessarily associated with other signs of sexual maturity.

Gigantism and Acromegaly.

In some cases there is no obvious lesion of the glandular organs. Others are associated with hypertrophy or tumours of pineal and pituitary glands, testes and ovaries. Obesity and excessive muscularity occur in hyperplasia and tumours of the adrenal cortex. In a fourth group there is arrested mental development. On the other hand tumours may be present in these various regions without any sign of precocity or obesity. Probably the thyroid and thymus glands are also related in some way with physical and sexual development. Precocity is possibly dependent on hypersecretion, while defective secretion in some one or more of the ductless and accessory glands leads to sexual infantilism, dwarfism, muscular atrophy, myasthenia, general alopecia, and impotence or loss of sexual function.

**Obesity** of hereditary origin rarely shows itself in early life. Dietetic obesity may occur in infants, even the breast-fed, from an excess of



carbohydrates in the food, and is exaggerated by imperfect oxidation, as in anæmic girls at puberty. Excessive diet is a common cause. Yorke Davies (1890) reported the case of a boy weighing 117 lbs. at 5 years of age and height 4 feet. He ate all day long and was fairly intelligent. Giant fœtuses of 20-24½ lbs. have been recorded, generally overdue, stillborn, and fatal to the mother. In congenital obesity the child is large at birth; steadily fattens, apart from excessive appetite or sexual abnormality, and maintains good health. Some of the cases of obesity associated with imbecility are cretinoid, and improve on thyroid treatment. An excessive deposit of fat is not uncommon in idiots. Obesity is a stigma of degeneration, when associated with early menstruation and masculine aspect; with large mammæ, deficient hair and feminine aspect in the male; or with a tendency to giantism. Abnormal obesity occurs with both infantilism and precocity. The infantilism may remain permanent as regards stature, mental and sexual development. Possibly these cases belong to the group of Myxœdematous Infantilism.

Guthrie has added much to our knowledge of this subject. He distinguished precocious obesity from the other forms by the blotchy state of the skin. It occurs most often with precocious sexual development, occasionally without, and is generally due to hypernephromata, less often to ovarian tumours. In Guthrie's female patient no tumour was found. Curiously in both sexes the hypernephroma is almost always on the left side. The muscular type is seen in males, the child being of the "John Bull" or "Miniature Drayman" type. Out of 16 collected cases, 7 were examined post mortem and revealed; hypernephroma 4, pineal tumour 2, testicular tumour 1. The hirsuties and sexual development may be that of an adult. Premature sexual development accelerates growth and ossification. Three cases in one family were reported by H. B. Robinson (1902), one girl and two boys under 6 years of age.

*Premature Senility*, called by Hastings Gilford "*Progeria*," from a Greek word meaning "prematurely old," is a rare condition. One case was described by Hutchinson, at age 3½, and completed by Gilford. He died from syncope at 17 years of age. Another began during the first dentition and died from angina pectoris, aged 18 years. The boys were remarkably alike; of small stature and proportions, and with imperfectly developed clavicles, lower jaw and membrane bones of the skull. The face was wizened and emaciated, hair scanty and white, skin shrivelled, body devoid of subcutaneous fat, hands knotted and with conspicuous veins and tendons, voice piping, and gait and standing posture those of old age. Anatomically there were found atheroma and calcification of the arteries and valves, fibroid kidneys, shrivelled adrenals and atrophied intestines. Somewhat similar senile changes in the arteries and fibrosis of pancreas, thyroid, pituitary and mesenteric glands, and spleen, were found by Ransom in a woman, aged 27, who died from diabetes. The sex organs were



entirely undeveloped. Premature greyness of the hair has also been reported by Caldwell in a case of sexual ateleiosis, and probably Ransom's patient was of the same type.

**Gigantism.**—Simple overgrowth is an exaggeration of normal growth, and to a certain extent characteristic of different families. If excessive it produces giantism, and is due to delayed epiphyseal union in long bones. Apparently giantism is acromegaly in the course of growth; acromegaly being produced without giantism if the epiphyses have united. Acromegaly may exhibit a tendency to myxœdema in early stages and to exophthalmic goitre in later ones. Giantism seldom begins before the age of seven years. Giantism and acromegaly are often due to tumours or other affection of the pituitary body. Giants are weak mentally and physically, sexually undeveloped, and die from trivial causes under the age of 30, usually under 22. These cases are interesting in comparison with those due to thyroid deficiency. Congenital absence of the thyroid and sporadic cretinism produce dwarfing. Disease of the pituitary gland causes giantism if the epiphyses have not united, and acromegaly if they have. Enlargement of the pituitary body has been found in myxœdema and cretinism, and fibrous goitre in acromegaly. Therefore, the evidence favours the view that loss of pituitary function leads to late ossification, and loss of thyroid function leads to early ossification, while possibly the two factors may be sometimes co-existent. Tamburini found the thymus enlarged in 8 out of 11 cases of acromegaly with pituitary tumour. Furthermore, hypertrophy of the hypophysis cerebri follows castration and extirpation of the thyroid. The thyroid was enlarged in 5 out of 23 cases of acromegaly, atrophied in 5, normal in 11 (Woods Hutchinson).

*Hemihypertrophy*, a condition of obscure origin, gives the child the appearance of being composed of two halves, one much larger than the other. The difference in size may extend to the internal organs or be limited to one or more of the external structures. It is congenital and non-progressive. When a limb, usually the leg, is affected, the bones are generally involved. In one recorded case, limited to the left side of the head and face, the cause was a multiple neurofibromatosis. Chronic enlargement of one limb may depend on a nævoid condition or lymphatic obstruction, giving rise to hypertrophy of the cellular tissues. In McGregor's case the right leg was chiefly affected and the head not at all. He died in his twelfth year after amputation of the limb and the right optic thalamus was found enlarged, the pituitary body normal.



## CHAPTER XVIII.

### THE THYROID GLAND.

*Congenital Goitre — Acute Thyroiditis — Cretinism — Myxædema — Exophthalmic Goitre — Parathyroids — Thyroglossal Fistula — Hygroma.*

At birth the thyroid gland weighs 1-2 drs. and contains colloid material. Iodine, bromine and arsenic have been found in it by different observers. In childhood the lateral lobes are usually equal; sometimes the right is greater than the left; and in rare instances one lobe may be absent. The isthmus may be absent or fused with one or other lobe. The pyramidal process is present in about half the cases; it is attached to the hyoid bone, less often to the thyroid cartilage, to the isthmus or a lateral lobe, or bifurcated and attached to both lobes; and is rarely double. In 60 children under the age of 10 years, C. F. Marshall found the foramen cæcum absent in 23, represented by a slight pit in 28, and as a ductus lingualis,  $\frac{1}{4}$  in. long, in 9.

*Fœtal or Congenital Goitre* is commonly hereditary and occurs in goitrous districts. Either parent may be affected, but in many instances neither is goitrous. It is noteworthy in this connection that Halsted extirpated the thyroid of pregnant bitches, and found that in the full-time puppies the thyroid gland was enlarged. Many of the infants are premature, stillborn, or die soon after birth; males predominate. Several sporadic cases have been ascribed to the action of chlorate of potash given to the mother during pregnancy, with a view to preventing miscarriages. In these cases the mother was a non-goitrous multipara and had had numerous miscarriages, but had never before borne goitrous children. Many women have taken this drug without it affecting the child. A similar goitre has followed the administration of iodide of potassium to a syphilitic mother. Generally the chlorate was taken at the same time. Ballantyne found evidence of thyroid hypertrophy in the fourth month of foetal life. Most of these infants with congenital goitres, ascribed to chlorate of potash, have died within a few hours. In a case of Simpson's (1866) the tumour gradually shrank, and at 4 years of age the child seemed healthy. In goitrous districts, e.g., Gilgit and Chitral, endemic goitre is only occasionally congenital and rarely a cause of cretinism (McCarrison, 1908). The goitrous enlargement may be vascular, parenchymatous or adenomatous. The vascular type is most common; large blood vessels and sinuses



are present, the acini are foetal in character and contain no colloid, and no cysts are found. The adenomatous tumour is encapsuled in the substance of the gland, contains one or more cysts, and even cartilage at the early age of 2 months. Occasionally it is fibro-cystic. The parenchymatous type is the same as in adults.

The symptoms are those of a tumour in the neck and due to pressure. In mild cases there is only slight dyspnœa or hoarseness. Large tumours cause severe dyspnœa, asphyxia, cyanosis and retraction of the lower ribs. They have to be diagnosed from congenital cysts in the neck and enlargement of the thymus. Mild forms recover spontaneously; large tumours cause death from pressure on the trachea, œsophagus, blood vessels or nerves, unless relieved by surgical measures.

*Acquired Goitre* may be sporadic or endemic. Its characteristics are the same as in adults. It often dates back to infancy; is more common in girls and on the right side; of the parenchymatous type; and often gets well spontaneously. The treatment consists in change of locality, alkaline waters, syr. ferri iodidi and other iodides, inunction with iodine or ung. pot. iod., and operation.

**Acute Thyroiditis.**—Acute inflammation of the thyroid is due to injury or strangulation, or a sequel of some infection, e.g. vaccination, specific fevers, throat affections and parotitis. A local swelling is found, either to one side of, or over, the trachea. It moves on deglutition and may cause pain, hoarseness, cough, dysphagia and fever; occasionally some dyspnœa and cerebral congestion. The pulse rate is increased out of proportion to the fever. It subsides or terminates in abscess. Apply cold and incise if necessary; tracheotomy is rarely required.

**Cretinism.**—Cretinism, or congenital myxœdema, and myxœdema are almost identical in their pathology, but clinically present points of difference on account of the age-incidence of the disease. In cretinism thyroid function has not developed, while in myxœdema it is lost; and the older the child at the onset the less complete are the signs. Cretinism may be goitrous or non-goitrous, endemic or sporadic. The endemic type is associated with endemic goitre and deaf-mutism. It occurs in the valleys of mountainous districts and is due to some cause in the climate, soil, or water.

*Endemic Goitre* is an infective disease, and is present in the mother in almost every case of cretinism in goitrous districts. These goitres are deficient in thyroidin, and the mothers are apt to have tetany during pregnancy. The cretinism is due to defective maternal thyroid function and a toxic effect on the thyroid of the infant. Goitre is present in 75 out of 100 endemic cretins over 10 years of age, but endemic goitre in the child is rarely the cause of cretinism. McCarrison found that in all but 2 out of 88 cretins the goitre appeared subsequently to the cretinoid symptoms, and that only 17 per cent. under 10 years of age had goitre. The goitre is generally adenomatous and functionally inactive.



*Sporadic Cretinism* rarely affects more than one in a family. The thyroid is almost universally absent, occasionally small or cirrhotic, and rarely a cystic goitre. *Infantile or Juvenile Myxœdema* may follow local injury, thyroiditis or a specific fever. A post-natal infection in early life accounts for some sporadic cases. In the juvenile type the child develops normally, until some illness, and then in a few months growth stops and signs of cretinism appear. Thus, it may be spoken of as "*Acquired Cretinism*." Psychic factors, such as grief, fright, nervous shocks, injury, and impairment of nutrition from any cause, are exciting rather than causal factors in sporadic and in many cases of acquired cretinism; the true cause being latent congenital thyroid defect.

*Symptoms.*—There is no doubt that cretinism and myxœdema are due to deficiency of thyroid secretion or *Athyroidism*. In its mildest form it gives rise to delayed closure of the fontanelle, delayed growth, mental dulness and occasionally enuresis. Between this and the worst type of cretinism are many grades. Typical cretins are abnormally small, and backward in physical and mental development. Infants rarely give signs before 6 months of age, or not until weaned, because up to this time they obtain some thyroidin in the milk. An infant cretin has a wrinkled forehead, puffy expressionless face, broad short hands, subnormal temperature, and is undergrown. The characteristics of the disease are numerous. The skull may be normal; dolichocephalic, enlarged posteriorly; or brachycephalic. The fontanelle is widely open. The hair is short, coarse and scanty; it may be fine, silky and luxuriant in early cases. The complexion is muddy, with dusky flush on the cheeks; features doughy, thick, bloated and devoid of expression, or dull, heavy, sad and unchanging. The lips are thick, everted and mauve in colour; the tongue large, thick, and often kept partly out of a large half-opened mouth; dentition late and milk teeth long retained. The nose is depressed at the root, short, and spreads out enormously towards the alæ. The eyes are wide apart, squint common and nystagmus rare; the eyelids swollen and baggy. The skin is dry, rough, and of a parchment-like tint; it is redundant, causing puckering of the forehead and puffy folds below the eyes. The abdomen is protuberant and umbilical hernia frequent; sometimes there is extreme lordosis. The body is stunted; the limbs stunted and often crooked. The hands are spade-like, thick and podgy, short and broad, with square finger tips. Both hands and feet are blue and cold, so much so as to suggest congenital heart disease. The muscles are flabby and badly developed; the movements deliberate and few. Speech is delayed and words are scanty; voice guttural and monotonous in later life. Masses of solid œdema, forming "*fatty tumours*" or "*supra-clavicular pads*," develop. Constipation is present from the earliest life; the vital powers are low and the temperature subnormal; the child sensitive to cold. The mental condition is dull, stolid, lethargic and backward. Sometimes the child is unable to recognise anyone. Many are good-natured; some idiotic and



unable to speak ; a few destructive and vicious. They are heavy sleepers ; averse to bodily exercise or too weak for it. The sexual functions do not develop and the sexual organs remain undeveloped in the male, but not invariably so in the female. Beard, axillary and pubic hair are absent.

The *Diagnosis* of the condition in the early stages of life is of the utmost importance for it is very amenable to treatment. It is often diagnosed in error, and more often is not recognised for some months or years. In the breast-fed the symptoms are delayed and rarely obvious until 6 months of age. Macroglossia, apathy, constipation and backwardness are suggestive signs.

The *Prognosis* is hopeless if no treatment is adopted. Many die young and few grow up ; life has been prolonged to 40 or 60 years, without any improvement in the mental state as age advances. Death is commonly due to broncho-pneumonia, tuberculosis, infectious disease or diarrhœa. The effects of thyroid treatment depend on the duration of life before treatment. It is more effective in the sporadic than in the endemic type. The longer the disease is untreated, the less is the improvement. If treatment is begun during the first year of life, the patient may grow up a normal child. Even in late cases remarkable improvement takes place. The physical and mental conditions are usually proportionate, but one may be more backward than the other. Improvement takes place more quickly in the physical than the mental state, and may be limited to it. When the mental state is only slightly altered and treatment begun early, the outlook is good. One of the first indications of improvement, evident in 3-5 weeks, is the diminution in the size of the tongue. Children taking thyroid extract do not succumb so readily to infectious disease. The treatment is of little use for old cretins, except to improve the appearance and epidermal structures. Female cretins sometimes become pregnant and bear marasmic or hydrocephalic infants.

*Treatment.*—Give thyroid in small doses twice or thrice a day, and increase it until the maximum dose for the child is reached. Continue the maximum dose for some time, and then gradually reduce it to the minimum dose. The treatment must never be wholly omitted for long. The early signs of a slightly excessive dose are nasal catarrh (L. Williams), rapid pulse and vomiting. Over-dosage produces mental excitement, irritability, restlessness, sleeplessness, depression, headache, thirst, vomiting, urgent dyspnœa, obscure pains, palpitations, intolerance of heat, rise of temperature and loss of weight. For such symptoms keep the child in bed and omit the drug temporarily, or reduce the dose. There is no danger to the heart from overstrain, as in adults, although the cardiac muscles may atrophy, for the child's blood pressure is low. A slight rise of temperature indicates the proper dose. Give liquor thyroidei m. 1-5, thyroid tabloids grs.  $\frac{1}{2}$ -5, or iodothyrene grs. 10, three times a day, or from  $\frac{1}{6}$ - $\frac{1}{8}$ th part of fresh raw sheep's thyroid twice a week. One sheep's thyroid is equal to 100 minims of the liquor. A suitable dose for a child of 6 months is gr.  $\frac{1}{6}$ - $\frac{1}{3}$ ,



three times a day. The signs of improvement are that the temperature rises to normal and remains there; initial loss followed by gain of weight, improved intelligence and growth in height. The skin becomes normal and subcutaneous swelling disappears. The eyes are brighter and the face more intelligent, with a natural flush on the cheeks. The tongue is not protruded, the voice less guttural, and snoring ceases. The hair falls out, and is replaced by softer and more natural growth. The appetite is good, dentition active, bowels not constipated, abdomen smaller and hernia disappears. There is a marked change in 6 months. The child may grow an inch in the first month, and from 6-8 ins. in a year. The rapid growth may lead to the legs bending, when the child begins to stand and run. Lateral curvature is often present in cretins after the age of childhood, and is increased by rapid growth. The resulting deformities are similar to those of rickets and due to bone softening. If the treatment is stopped, a goitre may develop, for the gland is not always quite atrophied. Cretins must be kept warm, for they are very liable to catch cold; carefully fed, for they are apt to get diarrhoea; and guarded against infection, to which they are particularly susceptible.

**Exophthalmic Goitre** is very rare in children. Barret (1902) collected 42 cases under 15 years, the youngest was 4 years of age. Of 5 cases recorded since, one was a girl, 3 years old, in whom the disease followed whooping cough and was fatal in her seventh year (Batchelor). It is more common in girls. The symptoms are much the same as in adults. Tachycardia is often the first sign; sometimes tremor, goitre or exophthalmos, the latter may be absent. Tremor is uncommon but chorea is frequent. The child finds it impossible to keep still. Other symptoms include great irritability and attacks of passion, anæmia, debility, loss of flesh, sweating, attacks of vomiting and diarrhoea, headache and depression. Retraction of the upper lids (Stellwag), defective convergence (Mœbius), and delayed movement of the upper lid on looking downwards (von Græfe) are also noted. The goitre is generally of the vascular type, and present for a considerable time before other symptoms appear. Sometimes cardiac symptoms come on suddenly or in the course of a few days, e.g., after whooping cough and broncho-pneumonia. Headache and fatigue are noted early. On the whole the onset is more acute than in the adult. The frequency of the pulse is a fair measure of the progress of the disease. Some of these acute cases have recovered in from 1-6 weeks. Exceptionally it lasts from 2-4 years and ends fatally from exhaustion, vomiting or diarrhoea. The duration is less than in the adult. Death is rarely due directly to the disease. Apparently it is caused by hypersecretion of the thyroid, an excess of normal secretion. In adults the parathyroids have been found atrophied and the thymus persistent, sometimes enlarged. Myxœdema is a rare sequel. These cases are treated by rest in bed, open air, over-feeding, massage, cold compresses, hydrotherapy, faradic baths and



faradisation of the goitre. X-rays may be useful at the onset, rendering the gland smaller and harder, and reducing the pulse, tremors and nervousness. They should only be used on one side as they may cause complete sclerosis of the gland and myxœdema. Surgical treatment consists in partial thyroidectomy, ligature of thyroid arteries, or division of the cervical sympathetic between the middle and upper ganglia. Most adults get well without surgical intervention. The chief drugs given are cod-liver oil, arsenic, iodine, bromide, belladonna, iron, phosphoric acid and phosphates; and the serum prepared from sheep or goats after thyroidectomy. Iodipin or red oxide of mercury ointment (1 in 4) can be rubbed in locally.

**Parathyroids.**—These glands, 1-4 in number, are situated in pairs attached to the posterior surface of the lateral lobes, towards the lower and median border, close to the œsophagus and trachea. They are yellowish or brownish red, round or flattened, like lymph glands, often imbedded in fat and difficult to identify. In structure they consist of large cells with deeply staining nuclei, arranged in columns in a stroma of connective tissue and a fibrous capsule. They are very liable to hæmorrhage, especially at or after birth, and may form cysts which slowly disappear. Operations have shown that extirpation in animals and man is followed by tetany; but if some parathyroid tissue is left, the tetany is mild and of short duration. Hæmorrhages and hyperplasia of the gland have been found in tetany. The gland is essential to life; no animal has been kept alive after complete removal of it. It regulates the motor nerve function. Apparently they are not embryonal thyroids. But extirpation of the thyroid in man, monkeys and dogs causes acute myxœdema. In rabbits this does not happen for the parathyroids escape removal. This implies that the functions of the two glands are identical.

**Thyroglossal Fistula and Cysts.**—The thyroglossal duct or tract runs from the foramen cæcum at the base of the tongue downwards in the middle line of the pharynx. As the hyoid bone develops it cuts the duct into an upper and lower part. The upper part forms the lingual duct and becomes obliterated, except the foramen cæcum. It is rarely so patent that a probe can be passed downwards from the foramen. If unobliterated it may become distended with secretion, often sebaceous, forming a cyst which is variously named *lingual dermoid*, *lingual hygroma* or *hyomandibular cyst*. This projects under the tongue like a ranula, or submentally in the median line; it is lined by squamous epithelium. If it bursts or is opened, it leaves a fistula with an orifice, usually in the median line just below the symphysis, through which a fine probe can be passed up to the base of the tongue. It must be excised from the neck.

From the lower part of the thyroglossal duct the isthmus of the thyroid gland is developed. The cysts arising from this portion are named *thyroid dermoids*: *thyro-hyoid*, if associated with the second cleft; *crico-thyroid*, if associated with the third cleft. The cyst may reach as low as the



manubrium. It is lined by compound epithelium, sometimes ciliated, and may contain thyroid tissue. Cysts or fistulæ of this nature usually have a pedicle passing through the thyro-hyoid or crico-thyroid membrane. They are firmly attached to the hyoid bone, and resection may be necessary for perfect cure.

*Lateral Cysts* may be of the same type, may start in lateral diverticula of the different clefts, or be of lymphatic origin. They are sometimes called *Cystic Hygromata* of the neck, and may be dermoids, adenomata, etc. Sometimes the cyst increases in size on expiratory effort. Congenital cystic hygromata, unilocular or multilocular, may be situated in other places, such as the axilla.

*Branchial Fistulæ* open in front of the sterno-mastoid, above the left sterno-clavicular joint or bilaterally at the level of the crico-thyroid space. They terminate on the outer surface of the pharynx near the posterior faucial pillar.



## CHAPTER XIX.

### THE ADRENAL GLANDS.

The Adrenal Glands are rarely absent. Probably in such cases accessory ones have been overlooked. The weight is  $\frac{1}{3}$  that of the kidney in the newborn;  $\frac{1}{28}$  in the adult. The cortex is closely related developmentally to the genital glands. It is derived from the pronephros; from a part of the mesodermic epithelium covering the Wolffian body. The medulla is connected genetically with the sympathetic system, being derived from the same blastema. Its cells are chromophil ones, staining intensely yellow or brown with chromic acid and its salts. Chromatin or chromophil cells are widely distributed through the body in the newborn. These glands receive their blood supply from the aorta, the renal artery, and the arteries of the diaphragm.

*Functions.*—Death is stated by Biedl to follow experimental removal of the cortex. As it is impossible to separate it from the medulla the experiment is of little value. In some way the gland is connected with the growth and development of the sexual organs, and apparently the sexual function depends on the cortex. Excessive development of the clitoris has been found in conjunction with adrenal hyperplasia. Excessive development of the sexual organs, pubic hair and fat, has been found in association with hypernephroma; but may occur without hypernephroma. In cases of retarded sex development, osteogenesis imperfecta and some developmental errors in the nervous system, hypoplasia has been found. Possibly the cortex secretes some substance which neutralizes the toxic products of metabolism. It contains no pressor substance, though it may possess an internal secretion which assists in the elaboration of the pressor substance, adrenalin, secreted by the medulla. Adrenalin causes constriction of the arterioles by direct action; and increases the rate and energy of the heart-beat by its effect on the central nervous system. This pressor substance is probably formed also in the chromophil cells, called *Zuckerlandl's parasymphathetic bodies*, in connection with the sympathetic and in the pituitary body, possibly Luschka's gland, and the intercarotid gland. The anterior lobe of the pituitary body and the adrenal cortex have several points of resemblance. The pituitary yields a pressor substance and hyperplasia is associated with acromegaly.

Apparently the most important function of the adrenal glands is to maintain blood pressure. The diphtheria toxin seems to have a profound



influence on these glands, so that they are unable to maintain blood pressure sufficiently, and death may result. The internal secretion may be absent in Addison's disease and asthenia from prolonged wasting. Possibly it is diminished, causing partial inadequacy, in tuberculosis, neurasthenia and cyclic albuminuria. Acute insufficiency has been described in adults, almost always associated with adrenal hæmorrhage. Conditions in which it is increased and altered in quality require investigation.

*Affections of the Adrenal Glands.*—On account of its liberal and peculiar blood supply it is liable to hyperæmia and hæmorrhage, especially under high blood pressure, e.g. in fits. Infarcts are not uncommon. Suprarenal apoplexy in the newborn has been already discussed (p. 130).

*Adrenal Tumours* may be simple ; may be associated with metastases of the skull (Hutchison's type) ; with simultaneous sarcoma of the liver ; with precocious puberty.

Hutchison's type occurs in infants and young children. It starts with ecchymosis of the eyelids and exophthalmos, either spontaneously or after trauma. Anæmia and debility may be noted for some weeks previously. A growth appears in the adjacent temporal region, and enlarged lymph nodes in front of the ear and at the angle of the jaw. Pain in the head is frequent. Total blindness is rare, but optic neuritis may occur. Vomiting is rare in spite of extension towards the brain ; fever is usually absent and anæmia secondary. The size of the abdomen depends on that of the growth ; there is no ascites. The growth is unilateral, occasionally bilateral ; varies in size from a walnut to a child's head ; and has little tendency to invade neighbouring organs. On section it is smooth, greyish white or yellow, and mottled with hæmorrhages. Death is due to anæmia and cachexia.

Out of 15 collected cases all but 2 were under 3 years of age. Nine out of 13 were males. Structurally they are described as sarcomata : of small round cells, 4 ; round and oval cells, 3 ; lymphosarcoma, 2 ; medullary, 1 ; melanotic, 1 ; and sarcoma, 1 ; and hypernephroma, 2. In 6 the cranial bones and lymph nodes were alone involved. Metastases have been reported in the calvarium in all cases, ribs 4, spine 1, sternum 1, tibia 1, bone marrow and periosteum of several long bones 1, and once in the ovaries and parietal pleura, also in the liver and kidneys. Primary adrenal tumours without metastases in the skull rarely, if ever, involve other bones. In the early stages the swelling may be mistaken for a sarcoma of the orbit, but in this affection the pre-auricular glands are rarely enlarged.

*Simultaneous sarcoma* of the adrenal and liver has been found in the first weeks of life. There is a diffuse infiltration with small round cells. The liver is enormously enlarged, without ascites and usually without jaundice. Other varieties of tumour are very rare.

*Precocious Puberty.*—The relationship of tumours to precocious puberty is considered on page 200. It is most commonly of the obese type, in girls



of 1-8 years with large genitals, hirsuties, brunette type of skin, sullen disposition and dull intellect. Thirst, increased appetite and vomiting may occur, and sometimes hæmaturia. The tumours are hypernephromata and show adrenal structure. Probably there is an excess of adrenal secretion.

*Addison's Disease.*—Monti states that 11 out of 290 cases occurred in children. The main symptoms are emaciation, pallor, asthenia out of all proportion to the amount of wasting, and pigmentation of the skin. This pigmentation may be an early sign or appear quite late. It may be limited to the palate or distributed in the usual situations. Other symptoms are dyspepsia, intractable vomiting, profuse diarrhœa, apathy, headache, vertigo, convulsions; subnormal temperature, occasionally febrile paroxysms. It lasts for months, sometimes for years. It may be fatal in a few days with symptoms like those of peritonitis. Diagnosis is almost impossible in early stages. Grünbaum states that adrenalin by mouth raises blood pressure in this disease, but not otherwise. The autopsy may reveal tuberculosis, total or partial, of one or both glands; conversion of the glands into scar tissue; tumours or hyperplasia. The glands sometimes appear healthy. The disease is caused by affections of the glands, or the cœliac ganglion and sympathetic nerves which control their secretion. Treatment so far has proved unavailing.

*Acute Insufficiency* of the adrenals sometimes occurs. R. S. Lavenson (1909) classifies cases according to the clinical symptoms into groups: (1) Sudden onset with epigastric pain and tenderness, followed by abdominal distension and death in a few days. (2) Profound asthenia and death within a few days. (3) Convulsions, coma and delirium or a typhoid state. (4) Sudden death, usually due to hæmorrhage. (5) Purpuric rash or hæmorrhage into abdominal viscera. Some cases present symptoms of more than one group.



## CHAPTER XX.

### THE MOUTH AND JAWS.

*Structural Defects—The Tongue—Stomatitis—Uvula—The Teeth and Dentition—The Salivary Glands.*

Apart from deformities, disorders of the mouth are commonly due to microbial infection to which it is unduly liable, because of the delicacy of the mucous membrane and the absence of saliva in the early months of life. At all ages organisms are found which become virulent under suitable conditions. Thus, the micrococcus of sputum septicæmia, staphylococci, diplococcus pneumoniæ and the diphtheria bacillus may be present in the saliva of children apparently healthy. The oral secretions are not bactericidal nor is the cavity sterile, but the harmless microbes present crowd out the pathogenic ones.

*Care of the Mouth.*—It is frequently recommended that the infant's mouth should be wiped out gently after each feed, with a soft rag dipped in clean water or weak boric acid solution. This is quite unnecessary and a common source of injury to the mucous membrane. The teeth should be cleaned twice a day. During illness the lips are apt to be dry, cracked and covered with sordes; and the tongue parched, furred and cracked. Benzoated lard or cold cream should be applied to the lips. For cleansing the mouth and removing fur from the tongue, swab hourly in bad cases with lemon juice, glycerine and water, glyc. ac. borici., boroglyceride 1 in 40, or a solution of boric acid or Listerine. Another useful mixture is sod. bicarb. grs. 10, sod. bibor. grs. 10, glycerin. dr. 1, tinct. myrrhæ m. 1-10, water to 1 oz. As antiseptics salicylic acid and benzoic acid solutions, 1 in 200, are the best. Eucalyptol and thymol are mere deodorants. Bicarbonate and biborate of soda are used to dissolve mucus; weak sulphurous acid for the destruction of moulds; liquor sodæ chlorinatæ or chlorine water for foetor and ulcers. As disinfectants and deodorants use, for douching, permanganate of zinc gr.  $\frac{1}{10}$  in 5 oz. of water; peroxide of hydrogen, 2 per cent.; lysoform, 0·5 per cent. For more constant action formamint tablets can be sucked frequently; each contains formic aldehyde gr.  $\frac{1}{8}$ ; or lozenges made of thymol 0·20, alcohol 2·00, sugar 200 gms., flavoured with essence of peppermint and divided into 200 parts. Parasitic growths and ulcers are best treated by painting with solution of silver



nitrate, 2 per cent., pure carbolic, copper sulphate grs. 2 to 1 oz., zinc chloride grs. 20 to 1 oz., tincture of iodine, or boric acid 10 per cent. solution.

*Deformity of the Jaws in relation to Suckling.*—The tongue is large and muscular, and the cheeks contain pads of fat immediately over the buccinator, bulging inwards and known as “sucking pads.” The under surface of the upper lip is somewhat papillated and the levator labii superioris well developed. These structures are utilised in the act of suckling, in which the movements are analogous to those adopted in milking cows and involve compression rather than sucking. The baby takes into its mouth the nipple and a portion of the areola, a cone-shaped mass, the base of which it squeezes between the palate and the lower jaw by elevation of the lower jaw, thus forcing out the milk from the distended ampullæ. The tongue is partially protruded over the lower gums, but the cheeks remain passive and are not drawn in as in suction. In ordinary bottle feeding, especially when the teat is small and like the so-called dummy-teat or “comforter,” the process is almost entirely one of suction. This is said to produce evil mechanical effects on the jaws and teeth.

To this have been ascribed irregularity of the teeth, deformities of the hard palate and dental arches, mouth breathing, nasal obstruction and deviations of the septum. The child constantly has its mouth partially open. Though the jaws are closed, the upper and lower incisors do not meet and leave a gap big enough to admit the tip of the little finger; and the upper incisors overlap the lower. The palate is high, narrow and arched, and causes contraction of the nose and nasal fossæ. The arch of the lower jaw may be narrowed; the teeth are crowded. These children often suffer from adenoids and large tonsils. It is a moot point whether the deformities are primary or a secondary result from continued sucking, the constant pressure by a hard teat and atmospheric pressure; or whether they are due to the adenoid and tonsillar hypertrophy. They have increased in frequency during recent years, coincidently with the increase in bottle-feeding and use of comforters. In races where breast-feeding is the rule, these deformities and the occurrence of adenoids are distinctly rare. It is more probable that the deformities are due to adenoids, for they are frequent in the breast-fed. If they were due to bottle-feeding they should be much more prominent in infants, whereas they are rare before the age of 6 years. Moreover, all these deformities may occur independently of each other. It is important that a proper cone-shaped nipple should be used in bottle-feeding and comforters forbidden, for undoubtedly the act of sucking may help to produce deformity in infants predisposed to it by the presence of adenoids. The relationship of these deformities to nasal obstruction is considered more fully in a subsequent chapter.

*Hare-lip and Cleft Palate.*—A hare-lip, whether median or lateral, should be operated on about the end of the first month of life. The sooner



it is done, the better is the subsequent development of the mouth and nose, and the closer is the approximation of the edges of the cleft in the alveolus, if present. The voice will develop normally, if there is no defect in the alveolus, but even a small notch in this may cause lisping.

The operative treatment of cleft palate depends partly on the extent of the cleft. If it is limited to the *soft palate* and there is no other defect, articulation will become perfect, provided the cleft is cured before the child acquires bad habits. It should be closed about the end of the second or beginning of the third year of life. In early infancy the parts are so small and the tissues so friable that the operation is difficult, immediate results may be bad, and the new soft palate small and imperfect.

If the cleft is in the *hard palate*, articulation will never be quite perfect, no matter how early the closure is effected, for there is associated defect in the nasal chambers. Articulation does not depend entirely on perfection of the velum. The voice is somewhat the same as that of a child with a high palate encroaching on the nasal chambers. Such a voice may be independent of palatal defect and be due to defective co-ordination of palatal muscles. On physiological grounds the cleft should be closed as soon as possible. Arbuthnot Lane recommends the fifth week as the best age, while other surgeons prefer to postpone operation until the third to the sixth year. It should be done in the fourth year by preference, or in the third year if the child is strong and healthy. Bad habits of speech are rarely learnt earlier. If done early, the naso-pharynx is exposed sooner to the mechanical factors on which its proper development depends, and the voice is said not to become nasal; the child is healthy; the operation is easier because of the absence of teeth and the possibility of obtaining a greater flap, and the tissues repair well. The child is more tractable, and the general health is improved because food can be taken better. Against the early operation, it may be urged that the results are as good or better at a later age; that a nasal voice will occur in any case if the cleft is in the hard palate; that the risk of death from shock and the risk of sepsis are greater, unless the teeth are carious; that the tissues are more lacerable and liable to slough; that the late operation is easier because there is more room and more tissue to deal with; and that older children are kept quiet more easily after the operation. Certainly the operation on the soft palate at a very early age is often unsatisfactory, being followed by much fibrosis and subsequent contraction. Undoubtedly the risks are less in the later operation and the results as good, if it is done before the fifth year. It is always important to train the voice afterwards, especially in saying "S" and "TH."

If the cleft is very large it may be better to operate early, and to adopt Brophy's method of forcibly approximating the maxillary and palate bones by wire sutures. By this means the halves of the velum are brought close together and can be adjusted without tension. It should



be done 10 days to 3 months after birth; after that the bones are too fully ossified to be displaced by a safe degree of violence. The hard palate should be closed before the soft, and the hare-lip operated on subsequently. It is a severe operation.

Cleft palate interferes with suckling. A teat with a flange on each side to fill up the cleft, may enable the child to take the bottle. If not, it must be fed by a spoon or nasal tube.

*Alveolar Defects.*—Total necrosis of the inferior maxilla has followed on alveolar abscess and noma. Partial necrosis of the alveolar border of both upper and lower jaw may occur from inflammatory and septic infections of the mouth. In one child an attack of measles was followed by this type of necrosis on the left side, with separation of the bone and the exposure of the permanent teeth, which subsequently dropped out. Cicatricial bands may be caused by ulcerative stomatitis or cancrum oris and produce closure of the jaws.

*Micrognathia*, or smallness of the lower jaw, produces a bird-like facial aspect. The prominence of the chin is lost and the upper incisors project. In unilateral cases the hyoid bone and the larynx may be displaced to one side. Opening the mouth, mastication and speech are interfered with, especially if ankylosis exists. Congenital cases are rare, and generally associated with developmental defects incompatible with life. The condition is due to premature ossification of the lower jaw on one or both sides, or to ankylosis from excessive development and altered direction of growth of the coronoid process and great wing of the sphenoid. The acquired variety is caused by interference with the growth of the epiphyses, or irregular nutrition of one or both halves of the jaw. Hence, it may result from injury at birth or subsequently, and from secondary necrosis due to periostitis or osteomyelitis, cancrum oris and abscess in front of the ear. Surgical treatment is of little value as regards appearance. Resection of the coronoid process cures ankylosis and osteoplastic resection of the lower jaw offers some hope of improvement.

The mouth may be abnormally large, *Macrostoma*; abnormally small, *Microstoma*; and rarely there is unusual hypertrophy of one or both lips, *Macrocheilia*, commonly nævoid or due to lymphangiectasis.

**The Tongue.**—Dryness of the tongue may be due to absence or deficiency of saliva from defective secretion or blockage of the parotid ducts. It may result temporarily from fear or it may be due to mouth breathing. The fur on the tongue is formed by proliferation of the epithelium and is increased by fever. Its appearance is modified by the lack of attrition and cleaning of the tongue by food. A tongue always becomes furred when the child is fed on liquid diet, partly because of the diet, partly because of the illness which renders such a diet necessary. In addition the tongue is modified by congestion and inflammation. Glossitis is usually



part of a general stomatitis. It may be caused by mechanical injury, sharp teeth, and irritants.

The tongue may be dotted or stippled; heavily coated or plastered with a thick paint-like coat; strawberry-like, due to prominent red papillæ on a white background; furred or shaggy; dry, brown, and encrusted; red, smooth and dry, denuded of epithelium (*Raspberry Tongue*); or cleaning, beginning at the tip and sides.

The *Geographical Tongue* (Annulus Migrans, Glossitis Areata Exfoliativa) is greyish white from hyperplasia and red from denudation of epithelium. It is generally seen at one to four years of age and is possibly a sign of the exudative diathesis. A greyish white spot appears on the edge of the tongue and extends on to the dorsum. It spreads by irregular concentric rings and becomes reddish in the centre. There is no ulceration. The rest of the tongue may be coated. Sometimes the patches are multiple and coalesce. The course is chronic, the results negative, and treatment unnecessary. It can be painted with strong chromic acid and then washed with alum lotion.

*Tongue-tie* is unimportant but, if the frænum extends right to the tip, it may interfere with suckling and affect speech subsequently. It can be snipped with a pair of scissors, taking care to keep close to the tongue and do too little rather than too much. In rare instances the tongue is adherent to the floor of the mouth by easily separable adhesions.

*Lip-tie* is a similar condition, due to an abnormally short frænum of the upper or lower lip. *Sublingual Ulcer*, at the junction of the frænum with the floor of the tongue, is generally due to whooping cough or other spasmodic cough, and occasionally to the friction of the lower incisor teeth in infants. Rarely a *Sublingual Fibroma* grows from the frænum in infants and may ulcerate. It is probably due to irritation by the incisor teeth. *Macroglossia* is due to hyperplasia, mainly interstitial. It interferes with or prevents swallowing. The tongue is unduly large, protrudes from the mouth, and may become eroded and fissured. It is seen in cretins and less often in mongols. Muscular macroglossia, lymphangioma, nævus, tumours, and gummata are rare. A few cases of macroglossia, due to neuro-fibromatosis, are on record.

**The Mouth.**—*Erythematous Stomatitis* in the newborn is the name given to the bright red appearance of the mucous membrane of the mouth and tongue present at birth. *Aphthæ in the Newborn*, or *Bednar's Aphthæ* (Ulcers Pterygoidea), were described by Bednar, in 1850, as only seen in babes from 2 days to 6 weeks of age, on the hard and soft palate. They are usually bilateral and symmetrical, one on each side of the median line or united by a bridge and butterfly shaped. At first there is injection of the mucous membrane; next a superficial epithelial necrosis, forming a greyish or yellowish white adherent secretion, which is soon exfoliated and leaves a superficial ulcer. These yellowish white patches are oval in shape and



surrounded by a red areola. They are due to rubbing and mechanical cleansing of the mouth after birth, and even before feeding, for they may be found before the child is put to the breast. If infected with pyogenic organisms, they may give rise to sepsis. *Epithelial Pearls*, milia of the mouth, are masses of epithelium on the gums and hard palate, minute, and milky-white or yellowish in colour. They undergo absorption, but may ulcerate. Retention cysts of mucous glands may also ulcerate. Both these conditions may be mistaken for Bednar's aphthæ. Treatment consists in painting with 2 per cent. silver nitrate solution.

*Thrush* or *Parasitic or Aphthous Stomatitis* is due to the *oïdium albicans*, *mycoderma vini* or the *saccharomyces albicans*. The fungus can easily be found by microscopic examination. It is rare in the breast-fed, most common in the first few months of life, in the bottle-fed, and under bad hygienic surroundings. Prolonged illness and malnutrition are important predisposing causes. The growth of the organism is favoured by dryness of the mouth, and its acid reaction and undue cleaning. The infection is usually derived from milk, and is spread by infected teats, comforters and spoons. Associated with more or less catarrhal stomatitis, small whitish patches of fibrinous exudation are seen on the tongue, lips, cheeks and gums. At first they are discrete, more or less circular, and look like flakes of curdled milk. They are apt to coalesce into larger patches, sufficiently extensive to cover the whole buccal cavity and fauces, and extend to the nasopharynx and œsophagus, occasionally to the larynx and stomach. Penetration of the epithelium by the fungus makes them adhere to the mucous membrane, and leave raw bleeding surfaces if forcibly detached. Partial exfoliation results from epithelial proliferation. After a day or two they become drier, yellowish and less adherent; and finally brownish and drop off, leaving normal mucous membrane. They often appear and recur in the debilitated, and are a sign of cachexia. Thrush gives rise to malaise, fever, enlargement of the submaxillary lymph glands, anorexia, wasting, and intestinal catarrh. Irritating intestinal discharges produce soreness and ulceration of the buttocks, and have given rise to the saying that "the thrush has gone through the child." In rare instances it causes general infection, with metastases in the form of embolic abscesses, perhaps due to mixed infection. The quickest cure is to paint the patches with 2 per cent. nitrate of silver solution. Boric acid, 10 per cent., is also efficacious. The child may suck a formamint tablet, crushed and put in a stout muslin bag, at frequent intervals.

*Perlèche* (pour lèche) is so-called because of the tendency to lick the red and rough affected angles of the mouth. Later on, radiating cracks appear and fissures, which are covered with exudate, are painful and bleed readily. They usually heal without forming scars. The disease chiefly occurs in school children, and is probably due to infection spread by pencil sucking. It may simulate *mucous plaques*, but it does not affect the inner



surface of the cheeks or cause deep ulceration, and is localised. It is treated by Friar's balsam, silver nitrate, tincture of iodine or antiseptic drying powder. Eczema of the lips and herpes labialis are simple and common affections.

*Catarrhal Stomatitis* occurs at any age, but is most frequent in the first year of life. It is usually secondary to dentition or local conditions and general disease. The mouth is hot and patches of intense redness are scattered over the swollen mucous membrane, especially on the tongue and gums. The child is fretful and restless, suffers pain on eating, and there is much salivation. The tongue is usually furred on the dorsum. After a time the epithelium necroses and forms yellowish patches, passing into the type of true aphthous stomatitis. Most inflammations of the mouth are preceded by the catarrhal form of stomatitis. In its mildest type simple desquamation occurs, and the tongue and gums especially remain intensely red.

True *Aphthous* or *Maculofibrinous Stomatitis* is most common at the crawling age. Small, discrete, yellowish white spots, subepithelial, with a reddish inflammatory areola, develop as a further stage of catarrhal stomatitis. The epithelium is rubbed off, and the fibrinous deposit rubbed off or absorbed, leaving a shallow ulcer with a red zone which soon heals. It usually comes on suddenly with fever and causes anorexia, burning pain, irritability, sleeplessness, and occasionally diarrhoea and submaxillary adenitis.

*Herpetic Stomatitis* may result from digestive disturbance and is not uncommon in the course of marasmus. Typical outbreaks, affecting the anterior end and the sides of the tongue, and the inner surfaces of the lips, occurred in a child aged 26 months, who had been ill for 18 months with recurrent ileo-colitis and marasmus.

*Membranous Stomatitis* is due to various organisms such as the diphtheria bacillus, pneumococcus, staphylococcus aureus and, rarely, the streptococcus pyogenes. Occasionally a whitish adherent membrane forms on the mucosa in debilitated children recovering from measles or whooping cough. The membrane is very like diphtheritic membrane. The tongue is rarely attacked. Impetigo of the face is almost always present, and the staphylococcus aureus can generally be found. The pneumococcal variety, also liable to be mistaken for diphtheria, affects the lips, gums, cheeks, throat and tonsils. Gonococcal infections occasionally occur, even in the newborn.

*Ulcerative Stomatitis* is a molecular spreading necrosis, with small cell infiltration at the onset. A spirillum and the bacillus fusiformis are often present. It occurs at 4-14 years of age, especially at the period of the second dentition. The chief causes are carious teeth; infectious disease, notably measles; insanitary surroundings; overcrowding and bad diet. It may be endemic in institutions. A similar state of the gums may occur



in mercurialism, scurvy, purpura and leukæmia. It depends on the presence of teeth, and almost always begins on the gums of the lower jaw, especially round the lower incisor or canine teeth. The outer surface is more affected than the inner. Swelling and redness are followed by necrosis of the mucous membrane, bleeding and purulent exudation, with fœtid odour and salivation. The teeth may be buried in the gums or the roots denuded, and become loose and drop out. The tongue is thickly furred, swollen and indented by the teeth; the lips and cheeks also may be swollen. The ulceration is liable to extend to the side of the tongue, lips, cheeks, and occasionally the tonsils and soft palate, and may progress on to necrosis of the alveolus and even the entire jaw. The constitutional symptoms are pallor, fever, irritability, depression, severe mouth pain, anorexia, inability to take food and toxæmia. Most cases recover in 7-10 days. The chief complications are adenitis, abscess in the glands and rarely in the tongue, Angina Ludovici, necrosis of alveolus or entire jaw, general sepsis and noma.

The general treatment of these mouth affections is on the lines mentioned under "Care of the Mouth," with the administration of chlorate of potash internally and diet of a liberal type. The mouth in ulcerative affections must be washed out frequently with Condy, permanganate of potash or permanganate of zinc lotion, and the ulcerated parts painted with tincture of myrrh 1 part, glycer. boracis 2 parts.

**Cancerum Oris**, *Noma* or *Gangrene of the Mouth*, is a specific necrotic process peculiar to children. It is never primary, but generally follows measles (50 per cent.), scarlet fever, typhoid fever, and occasionally other fevers. It may follow diphtheria and ulcerative stomatitis, and I have seen it, in a girl of three, as a sequel of acute gastritis. I am inclined to think that the administration of mercury predisposes to the infection. To a certain extent it is infectious or mildly contagious, and may break out in epidemic violence in institutions as a sequel of epidemic measles.

It may be defined as a mycosis developing on a specially acquired predisposition. The filaments spread deeply into healthy tissue and give rise to coagulation necrosis, analogous to hospital gangrene but without the production of gas. Primarily it is a simple infection, though in its later stage it is a mixed one. Decayed teeth predispose, through setting up gum conditions favourable to the growth of the specific organisms.

*Bacteriology*.—A long and thread-like leptothrix is often found. Walsh (1901) thinks two organisms are necessary, one to cause primary necrosis; and the other a saprophyte, to produce putrefaction. He found diphtheria bacilli in 8 cases, once in pure culture; 4 after measles and 4 after ulcerative stomatitis. Herrman (1905) ascribes it to two different organisms; one the "fusiform bacillus," commonly found in Vincent's



angina, identical with the spirillum sputigenum of Miller and corresponding to the streptothrix of Seiffert-Perthes; and the other the spirochæte dentium of Miller. These organisms are believed by Herrman to be different stages of the same, and he has named it the Spirochæte of Necrosis. Both are motile; normally present in the mouth, except in toothless infants; much increased in number by hyperæmia of the gums, in gingivitis, in ulcerative and gangrenous stomatitis, different stages of the same process, and in noma of other parts. The organism is not a bacillus and cannot be grown or stained in the usual way. It is best stained by dilute carbol fuchsin, and is decolourised by Gram. It requires very favourable conditions for its development, and consequently noma is only slightly contagious. The arguments in favour of this organism being a causative agent, are that it is present in very large numbers or in nearly pure culture; that it gradually disappears during healing; that very few other organisms are present; and that it penetrates into apparently healthy tissue beneath the necrotic layer. On the other hand it has a very wide distribution, cases are relatively few, and it is possible that necrotic tissues afford a suitable soil for its development. Though it may be the common organism, it is probable that other organisms may be important agents in some cases.

The primary lesion is an inflammation of the gums, and the presence of teeth appears to be essential. The tongue, cheeks and tonsils are generally affected secondarily. In rare instances it begins on the surface of the cheek and extends inward, on the ala nasi or at an angle of the mouth. It may affect the ear or vulva. It is generally, not invariably, unilateral. Frequently it begins as a bleb or a black spot on the inner side of the cheek, but the bleb is not often seen as the cheek rarely attracts attention at this stage. Sometimes slight ulceration of the mucous membrane is found first with a surrounding of intense hardness. The cheek is swollen, shiny and of a waxy pallor, not very tender or painful, and feels indurated. This stage is followed by rapid ulceration and extensive gangrene from within outwards. The skin of the cheeks becomes very tense, red and shiny, then livid and black. An external bleb often forms; the black patch becomes a slough and perforation ensues. As it breaks down it becomes very offensive and the fœtor is intense. The necrosis extends in various directions and a huge excavation is formed. Occasionally the parts become emphysematous. Hæmorrhage is rare for the vessels may be penetrated by the filaments and are plugged with clot. These cases terminate in 80-90 per cent. fatally from sepsis, secondary infection or septic pneumonia. In a few the process is arrested, sloughing ceases and rapid healing ensues, but more or less deformity from cicatricial contraction remains. Teeth drop out and there may be necrosis of an alveolus, or of the superior or the inferior maxilla.

*Treatment* must be stimulating; plenty of nutritious food; carbonate of ammonia, strychnia and cinchona, and brandy. If it is not too extensive,



excision and free cauterisation with Paquelin's cautery is the best treatment. In other cases, dry with lint and rub nitric acid or pure carbolic with sticks into the edges of the slough after cutting away the sloughs and sequestra. During the process protect the sound skin, applying nitric acid several times and drying after each application, and finally dust with iodoform or some such antiseptic, and smear the surface well with carbolic oil. Milder measures of treatment are painting with tincture of iodine or 10 per cent. silver nitrate solution, and douching frequently with 2 per cent. hydrogen peroxide.

**Uvula.**—The uvula may be bifid, unduly small or hypertrophied. If very large, it is liable to cause troublesome spasmodic cough, gagging, and even attacks of dyspnœa. A portion may be cut off. It is frequently swollen and œdematous in inflammatory affections of the mouth and throat, and rarely as a limited disease. Diphtheritic patches may be seen on the surface, but membrane in this situation is not necessarily diphtheritic. Superficial ulceration is not uncommon. A rare case of calculus has been reported in a baby of 2 months old (Goodall, 1898) in the form of a white globular mass on the anterior surface; it produced dyspnœa from nasal obstruction on lying down. In structure it was probably a degenerated mucous gland. A few similar cases of calculus of the soft palate have been recorded in adults.

**The Teeth and Dentition.**—In rare instances the child is born with one or more teeth already cut, a condition called *Dentitio præcox*. It may be a family peculiarity. The teeth cut are generally the lower central incisors, occasionally the upper ones, and rarely the molars. As many as nine were present in one case. They are usually loose, fall out or are pulled out, and are not replaced until the second dentition. Sometimes they are supernumerary teeth and replaced by milk teeth; occasionally they remain fixed. They are generally small, ill-developed, with thin and imperfect enamel and perhaps no fang. Premature dentition is due to the premature occurrence of the process leading to teething; to excessive development of the teeth or an atrophic condition of the gums; possibly to inflammation in the dental follicles or intra-uterine stomatitis; and may be associated with other malformations and anomalies, such as cleft palate. They are not an indication of intelligence, although they have been recorded in the past in Julius Cæsar, Richard III, Louis XIV, Mirabeau and Mazarin. If they are loose or giving rise to trouble, they must be removed. They may interfere with suckling, injure the nipples, or cause ulceration of the under surface of the tongue.

**Milk Teeth.**—The 20 temporary milk teeth are cut in the following order:—lower central incisors at 5-9 months; upper central and lateral incisors, at 8-10 months; upper molars, lower lateral incisors and lower molars at 12-14 months; canines at 16-22 months; and the second molars at 20-30 months. Or a more simple table may be given as follows:—



Average.		Date of Eruption of Milk Teeth.	Range.
6 months	..	Lower Central Incisors ..	6- 9 months.
9	„	Upper Central Incisors, and Upper Lateral Incisors ..	8-12 „
12	„	Upper Molars, Lower Lateral Incisors and Lower Molars .. ..	12-15 „
18	„	Canines .. ..	15-24 „
24	„	Second Molars .. ..	20-36 „

The first tooth is a lower median incisor in 85 per cent., and an upper median incisor in 15 per cent. Spanton (1907) gives the average date of eruption of the first tooth as 237 days ; males 252, and females 221. The milk teeth drop out in the same order by absorption of the roots. Teething is due to absorption or atrophy of the superjacent gums, and is not helped by lancing. Sometimes teeth are cut unduly early for no apparent reason or from congenital syphilis. In the mouth at birth there are also 24 permanent teeth ; 28 at 3 months, and 32 at 3 years. Breast-fed children generally cut their teeth earlier than the bottle-fed, probably because the salts in human milk are more readily assimilated than those in the cooked milk of other animals. This is not invariably the case, for bottle-fed infants may begin teething in the fourth month of life. Delayed and irregular dentition are due to cretinism, rickets, and maldevelopment from numerous causes ( *Dentitio tarda* ).

*Abnormalities* include complete absence of teeth throughout life, an excessive or deficient number, displacement, malformation, irregular eruption and imperfect development. Supernumerary teeth are usually small, distinct, and in the upper jaw. In prognathism the lower teeth project in front of the upper. Thumb sucking forces the upper teeth outwards and the lower teeth inwards. Lip sucking forces the teeth inwards. Structural defects in the milk teeth are due to causes acting in utero. J. G. Turner (1907) has drawn attention to malformation of enamel transmitted through five generations by those affected, but not by the unaffected. The teeth were stunted and the enamel absent or defective. It especially involved the earlier teeth, and the third molars were normal. Hypoplasia, or imperfect development due to malnutrition, affects both sets of teeth, but especially the permanent ones. Of the milk set the canines and molars are most damaged, implying an early post-natal cause. Of the permanent set the central incisors, the parts developed during the first two years of life, are most imperfect. If the defects are limited, the cause may be a local one. Hutchinson's teeth are defective permanent ones. The upper central incisors are dwarfed, laterally and vertically, and along the biting edge are crescentically notched. The lateral incisors are peg-like, and the



first molars "dome-topped." He ascribed the malformation to syphilis; Henoch, to rickets.

*Dental Caries.*—The predisposing causes include heredity, giving rise to irregularity in shape and size, crowding, pits and fissures, and imperfect chemical structure; severe or long illness, exanthemata, chronic tuberculosis and toxic conditions in early life, causing flaws in the enamel. The portion of tooth formed at this time is discoloured, pitted and grooved; and there is abrupt transition to normal along a horizontal line. Diet may influence the growth of the tooth before the age of six years, for calcification begins about the end of the first year. Therefore deficiency of lime in the food is a factor. Diet also affects the external surfaces, e.g., acids and a lack of hard substances which clean the teeth. Early decay of the milk teeth is due to acid fermentation of food in the mouth, destroying the enamel and permitting penetration of the dentinal tubules by microbes, that is, it progresses from without inwards. Circular caries at the base is not uncommon and may lead to the tooth being broken off. It is not eventually basal if the tooth is only partly cut. The upper incisors and molars are most liable. According to Miller's Theory of Decay the first stage is decalcification or softening of the inorganic enamel, and secondarily dissolution of the softened residue. The decalcifying agent is lactic acid due to the fermentation of particles of carbohydrate lodged between the teeth. It, therefore, depends on the regularity of the teeth and the nature of the food, neglect of cleanliness, and lack of cellulose which in mastication acts as a cleanser. There is always solution of continuity of the enamel before the organic material of the dentine is attacked. Thus, caries is essentially microbic in origin. Starchy foods are particularly apt to stick in crevices, whereas sugars are soluble and quickly washed away.

*Disorders of Dentition.*—Although dentition is a physiological process and normally gives rise to no trouble or inconvenience, like most vital processes it may be disturbed. Its influence has been enormously exaggerated and many ailments, general, local and reflex, have been ascribed to it, although they are merely coincident. Rickets is the true cause in many instances, by virtue of its nervous instability and liability to catarrh. The gums may be hot, tender and swollen, and the child feverish, e.g., T. 105° F. It is probable that dentition does not cause fever, unless there is oral sepsis or pain induced by inflamed gums. Traumatism, due to biting teething rings, is a great cause of gum injury and gingivitis, in which the gums are dry, hot and covered with sticky mucus. Salivation is suppressed; stomatitis and adenitis may ensue. On account of the itching and pain in the gums the child is irritable, restless, refuses food, keeps putting its fingers in its mouth, wakes and screams with pain or anger, pulls and rubs the ears or back of the head, and is generally out of sorts. The temperature is very irregular and often raised for several days, only a few hours at a time. While feverish the child is liable to catarrh of the respiratory



passages and alimentary tract. Many children are said to "cut their teeth with bronchitis" or "with diarrhœa." Among the poor diarrhœa is almost synonymous with teething and is rashly neglected in consequence. The digestive disturbance is commonly due to the continuance of a diet as liberal as during the pre-febrile stage. Nervous irritability is frequent in the rachitic and the neurotic. Nervous symptoms, such as laryngospasm, spasmodic cough, muscular twitchings, tetany and convulsions, are often ascribed to teething. Fits are far more common before than during the teething age. They are, in my experience, most liable to coincide with the eruption of the canines, the age when the influence of rickets is often well marked. Otitis media and earache may be due to extension of inflammation, or more probably to adenoids than produced by teething. In all cases of disease during dentition the true cause of the illness may be overlooked. I have known acute cerebrospinal and post-basal meningitis, pneumonia, the onset of specific fevers, and many other serious and mild ailments wrongly put down to teething. Almost every variety of rash, especially those due to intestinal toxæmia, has been called a "*gum rash*" or "*teething rash*." We may sum up the effects of dentition as follows. It is painful if the gums are unhealthy. Fever is due to local inflammation or other causes. Secondary catarrh of the mouth may extend to the ears, lungs and alimentary tract. Bronchial catarrh may arise in this way or is secondary to catching cold while feverish. Diarrhœa and vomiting may result from inability to digest food in consequence of the fever. Irritability, fretfulness, restlessness and insomnia arise from local irritation, gingivitis, stomatitis, and gastro-intestinal disturbance. Convulsions are indirectly produced by teething, in consequence of imperfect digestion of food, colic, and the absorption of toxins from the intestines during fever. Thus, the disorders are due to local conditions, fever, and secondary reflex irritation acting on an unstable nervous system. Retraction of the head is sometimes due to the eruption of molar teeth. Reflex symptoms are most likely to occur in nervous children. In a boy, aged 7, local pain in the cheek was definitely due to caries of the lower molar. Facial eczema is sometimes worse during the eruption of a tooth.

*Permanent Teeth.*—The first molars are cut at 6, central incisors at 7, lateral incisors at 8, and the bicuspid at 10 and 11 years of age. The canines appear between 12 and 13, the secondary molars between 12 and 15, and the third molars or wisdom teeth after the age of 17 years. Sometimes wisdom teeth are never cut. The disorders of dentition at this period are those of local discomfort and inflammation. Ulcerative stomatitis and other mouth affections may be set up by irritation of the fangs of partially extruded milk teeth. Such symptoms as fever, restlessness, insomnia, salivation, reflex cough, earache and fits depend upon local secondary inflammation or are of intestinal origin, set up by bolting food on account of pain involved in mastication. Nervous symptoms already present



may be exaggerated. Fits may occur in the predisposed. Adenitis is not uncommon, but cancrum oris and necrosis of the jaw are rare complications. Caries is dependent upon local causes and constitutional debility. The early appearance of the first molars leads to them being mistaken for temporary teeth, and to caries being neglected. They begin to calcify in the twenty-fifth week of foetal life. They should be carefully watched, and may require removal, if the mouth is small, for crowding of the teeth is a great cause of decay.

*The Teeth and Nutrition.*—Good health and digestion depend on good mastication and for this good teeth are necessary. Up to the age of 10 years mastication depends on the temporary teeth, reinforced at 6 years by the first molars. During this period of life growth is very active, and it is important that the milk teeth should be maintained in as perfect a state as possible and not allowed to decay. Caries of the teeth causes imperfect mastication and bolting or refusal of food on account of pain. It may set up periostitis, alveolar abscess, necrosis of the alveolus, antral suppuration, oral sepsis, ulcerative stomatitis, cervical adenitis and susceptibility to tuberculous infection. Constitutional effects arise from the absorption of toxins from the mouth, or the swallowing of decomposing and septic material from decaying dentine and food retained in the cavities of the teeth. Thus are produced anæmia, stomach troubles and malnutrition, possibly headache and fits. The malnutrition and reflex irritation predispose to pavor, habit spasm, tremor and chorea. Caries is progressive and spreads from one tooth to another, and even from a milk tooth to a permanent one. An alveolar abscess round a temporary molar may loosen a permanent one by destroying the developing roots.

Serious illness and disorders of nutrition during the first two years of life affect the incisors, canines and first molars, in which calcification is proceeding. Calcification begins in the other teeth in the third year of life, and may be affected by illness then or subsequently, up to the time it is completed. Fevers, especially measles, do much harm. The enamel is thinned, pitted, grooved or honey-combed. Often a discoloured transverse furrow separates the affected from the healthy part of the tooth. Early caries is a common sequel. Rickets affects the permanent teeth by malnutrition of the dental germs, often due to local mouth affections; and crowding, from imperfect growth of the jaw. It is doubtful whether mercury, given in infancy, affects the permanent teeth, though it is said to damage the enamel of the first molars, incisors and canines. More probably the dental defect is due to malnutrition or local affections of the mouth.

*Management of Dentition.*—Teething rings do more harm than good. Keep the mouth clean and allow no dirty articles to be put in. Use a badger-hair gum brush twice a day. If there is diarrhœa, give castor oil, tincture of rhubarb and salol, with glycerine, tragacanth, and peppermint.



water three or four times a day. Bromides and tincture of opium can be added if necessary. An initial dose of Dover's powder, gr.  $\frac{1}{2}$ -1, and grey powder grs. 1-2, will cut short the attack. For nervous irritability and restlessness, especially if there is the least indication of muscular twitching, give bromide grs. 3-5, or phenazone grs. 1-2, three times a day. Opiates and teething powders are unsuitable, especially the latter, because of the uncertainty of their composition; they usually contain calomel and occasionally morphia. Brush tender gums with chlorate of potash grs. 10, glyc. boracis to 1 oz. If they are ulcerated add resorcin grs. 10-20. Local sedatives of laudanum, cocaine, etc., are better avoided. The use of the lancet is unnecessary, for there is no tension of the overlying gum, and there is no evidence that lancing assists eruption. The subsequent scar, if directly over the gum, might make teething more difficult. Local bleeding by scarification affords local relief and reduces the tendency to convulsions.

*Care of the Teeth.*—Clean teeth do not decay. The greatest care possible should be taken of them from the time of eruption. They should be cleaned twice a day, with a soft rag at first, and later on a soft tooth-brush and a mild dentrifice, such as prepared chalk or soap and warm water; a few crystals of permanganate of potash and a few drops of tincture of myrrh in a tumbler of water; or thymol grs. 2, ac. benzoic. grs. 45, tinc. eucalypt.  $\frac{1}{2}$  oz., ol. gaulther. m. 20, sp. vin. rect. ad. 1 oz., using a few drops in water. Milk teeth should be periodically inspected and the cavities filled up. In the early stages of caries it is not a painful process. By this means the teeth are preserved as long as possible and the second dentition will be more satisfactory. Useless decayed teeth must be removed, though the extraction of temporary molars may cause irregularity of the permanent teeth. Still greater care should be taken of the permanent set. Cracking nuts, biting thread, etc., are liable to injure the enamel. The teeth should be brushed after each meal.

*Toothache or Odontalgia.*—An examination by the British Dental Association showed that there were 37,000 decayed teeth in 10,500 children of an average age of 12 years in Poor Law Schools; 86 per cent. had caries. In 560 Public School boys of the same age 3,521 carious teeth were present, and 87 per cent. had caries. The only sign may be neuralgia. Frequently, the local disease is indicated by irritation of the pulp without exposure, and the tooth is very sensitive to heat or cold. If the pulp is exposed, heat or cold gives rise to more violent pain, often widely distributed, referred, and neuralgic. Other local effects are suppuration of the pulp under a stopping, periostitis, alveolar abscess or fistula, and necrosis of the root. Anæmia and submaxillary adenitis are common.

The pain is relieved by a hot solution of bicarbonate of soda; tannic ac. 1, sp. vin. rect. 5 parts, painted on the gums and round the teeth; a plug of cotton wool dipped in oil of cloves, camphor, creosote, laudanum, or chloroform; and Gregory powder or phenacetin internally.



**The Salivary Glands.**—The salivary glands are rarely affected in the newborn. From pyogenic infection both the submaxillary and the sublingual glands may become acutely inflamed and suppurate, discharging pus through the ducts or forming abscesses (*Sialo-Adenitis*). Congenital and progressive *Hypertrophy* of the sublingual glands has been reported. It gave rise to difficulty in nursing and a bilateral tumour at the base of the tongue, which was at first thought to be a double ranula. The tongue became enlarged, dysphagia increased, and the child wasted. On operation it was found to be due to congenital hypertrophy or chronic inflammation. A *Ranula* is a congenital or acquired retention cyst of this gland, usually bilateral. It forms a reddish, shining swelling under the tongue. Congenital cysts may arise in other glands, due to atresia of the ducts. Two cases of bilateral *Swelling* of the *Parotid* in the newborn have been reported. The swellings were soft, elastic, painless, and the duct apparently free; saliva was present. They gradually subsided in 4 weeks. Similar swelling might be due to occlusion of Steno's duct. Angioma, adenoma, sarcoma and tuberculosis are very rare. *Acute Parotitis* is due to iodism or mumps (q.v.).

*Secondary Parotitis* is due to infection from the mouth, viâ the duct, and occasionally from the middle ear, viâ the Glaserian fissure. Predisposing factors are lack of care in cleaning the mouth in infants after food and vomiting, damage done to the mucous membrane in this process, and mouth breathing. The common causes are enteric fever, scarlet fever, pneumonia, peritonitis, stomatitis, malnutrition, and operation or diseases in which feeding by mouth is temporarily stopped. It is non-contagious, usually unilateral, and is liable to suppurate, except in the mild form often seen in marasmic infants. In marasmus the swelling may be the only indication. More often it is ushered in by local pain and tenderness, and pain on movement of the jaw. The glands are swollen, firm and tender, chiefly in front of the ear. Swelling spreads to the overlying tissues, and the cheeks become brawny, the eyelids puffy and the lids swollen. It gives rise to general malaise, dryness of the mouth, anorexia, thirst, fever, headache and depression; sometimes giddiness and tinnitus. In 4 or 5 days the inflammation reaches its maximum. It then subsides gradually in about another fortnight or suppurates. Pus may be deep-seated and cause no superficial signs, or give rise to increasing brawniness, œdema, redness and heat of the skin, and a softened area about the centre of the gland. The patient is more ill, with irregular fever, perhaps chilliness, and shooting pains. As the abscess increases in size, it burrows into the surrounding tissues, discharges through the parotid duct or bursts into the mouth, external auditory meatus or through the cheek. The resulting fistula heals with difficulty. If an early incision is made, the pus is discharged and the wound heals in a few days. Death may result from septicæmia or from the primary disease. Necrosis of the jaw, destruction



of the temporo-maxillary articulation, and hæmorrhage or thrombosis of the facial artery or the internal jugular vein are complications. Facial palsy is the most common sequel.

Preventive treatment consists in oral antisepsis, sucking a rubber teat, and sialagogues. At the onset give a purge, and use dilute Condly as a mouth-wash. Apply cold compresses or paint with glyc. belladonnæ. If there is a suspicion of pus, surgical measures are necessary. An incision should be made on the fourth to sixth day, if the swelling increases and becomes œdematous or if the temperature is high and intermittent. Do not wait for fluctuation. Incise the parotid fascia freely.

*Salivation* is due to local causes and in rare instances is idiopathic, even as early as the third year. It is produced by inflammatory infections in or about the mouth and its glandular appendages; by reflex causes, such as nausea, vomiting, gastralgia, gastro-intestinal derangements; hysteria, epilepsy, hydrophobia and mania, some of which may act on the central nervous system; and drugs, such as jaborandi, pilocarpin, mercury, lead and antimony. Idiopathic salivation may set in early during dentition, perhaps suddenly, and continue indefinitely. It resembles a neurosis in its intermittent character and its tendency to be affected by psychic influences and diminished by emotion. Sometimes it only occurs during the erect posture and diminishes or ceases during sleep. In other cases there is a continual dribbling, night and day, and the clothes become saturated. It is either due to increased secretion or to failure to acquire the habit of automatically swallowing the saliva. In Jordan's cases (1897) both children had fed largely on porridge. The duration is a matter of uncertainty. It tends to abate with the development of the body and to yield to drugs. Iron is beneficial, though the affection is not due to the anæmia which may be present.



## CHAPTER XXI.

### THE THROAT, PHARYNX, AND ŒSOPHAGUS.

*Tonsillar Affections—Pharyngitis—Adenoids—Retro-pharyngeal and Retro-œsophageal Abscess—The Œsophagus.*

Lymphoid or adenoid tissue surrounds the posterior nares and fauces forming Waldeyer's Lymphatic Ring. It is aggregated into masses, viz., (1) The Faucial Tonsils; (2) The Lingual Tonsils, two small masses at the base of the tongue between the V of the calciform papillæ and the glosso-epiglottidean fossæ, tending to disappear at puberty; (3) Luschka's or the Pharyngeal Tonsil; (4) scattered patches, on the lateral the posterior walls of the pharynx, which give rise to chronic follicular or granular pharyngitis. The lymphoid tissue in the membranous part of the Eustachian tube is called the tonsil of Gerlach.

The lymph vessels from the tonsils run to the submaxillary glands, especially those at the angles of the jaw, and thence to the cervical glands. Those from the naso-pharynx run to the lateral pharyngeal glands in the bucco-pharyngeal fascia behind the tonsils, or into the small retro-pharyngeal glands, and thence to the deep cervical glands. Swelling of the deep cervical glands alone is a sign of naso-pharyngeal, not of tonsillar disease.

**The Tonsils.**—*Etiology of Throat Affections.*—Hereditary predisposition is often present. The rheumatic diathesis is a predisposing cause and an attempt has been made to differentiate a specific rheumatic sore throat. Children are very liable to catarrhal affections from lowered vitality, cold winds, chills and dusty streets, especially the irritating dust from wood pavements in hot weather. Many cases are due to various infective organisms, and may occur in specific fevers. The decomposition of retained secretions in pitted tonsils is a constant source of infection.

*Simple Tonsillitis* is a catarrhal inflammation. In its mildest form it is spoken of as a "relaxed sore throat." The onset of a severe attack is like that of an infectious disease. The tongue is furred, breath offensive, vomiting and diarrhœa common. In infants there may be convulsions. The voice is thick, mouth open, and breathing difficult. Pain varies with the severity, so too the constitutional disturbance and fever which is rarely above 104° F. Older children may complain of dysphagia. A deep red and somewhat glistening erythema of the tonsils is visible, and it often



spreads to the palate, fauces and pharynx. The submaxillary lymph nodes are enlarged and painful. Recovery is rapid.

*Follicular or Lacunar Tonsillitis* is common in children, especially those with enlarged tonsils, and infrequent in infants. It is primarily a microbial inflammation of the crypts or follicles, and the glandular tissues are involved secondarily. The follicular variety is supposed to be a primary inflammation of the lymph follicles, and the lacunar variety is a more severe form, either general or limited to the crypts. The distinction is unnecessary, for it is doubtful that the lymph follicles are ever primarily involved. The use of the word "follicle" for "crypt," as well as for a collection of lymph cells, is confusing.

The onset is acute and may suggest possibilities of a specific fever or pneumonia. Sometimes the child seems little ill and has no local discomfort. More often there are found general malaise, headache, muscular pains and a temperature of 101-105° F. Anorexia, furred tongue, nausea, vomiting, dysphagia and constipation may all be present. Except at the onset the affection is almost invariably bilateral, and often associated with catarrh of the fauces and pharynx. The tonsils are enlarged, bright red, and covered with dots of whitish or yellowish-white secretion, projecting through the mouths of the ducts and composed of cheesy material, inspissated secretion, mucus or muco-pus, epithelial cells, debris of food, and micro-organisms. The secretion can be pressed out of the crypts or easily wiped away from the surface with a swab, leaving no ulcer and no bleeding surface. It may be aggregated by coalescence into a coherent membrane with no definite edge. The patches quickly re-appear after removal. The submaxillary lymph nodes are generally enlarged and tender. Even severe symptoms subside in 24 hours. Fever and malaise rarely last more than 2 or 3 days and the exudation disappears in another day or two, the swelling of the tonsil subsiding rather more slowly. Occasionally chronic follicular tonsillitis is present without any symptoms. The tonsils are more or less enlarged and the crypts invariably blocked with secretion.

The younger the child the more likely are the throat symptoms to be inconspicuous or absent, and the affection to be overlooked. Pain is often absent. Older children may complain of soreness, pain on swallowing and pain about the angle of the jaw. It is not always possible to exclude scarlet fever until sufficient time has elapsed to permit the appearance of a rash. A blotchy erythema is not uncommon, but differs in characters and distribution from the scarlatinal rash. The appearances of the fauces are sometimes suggestive of diphtheria. Cases may be wrongly diagnosed because the throat is not examined.

*Septic or Ulcerative Tonsillitis* differs from the previous variety in being more virulent and not limited to the tonsil. It is usually unilateral at the onset and often extends to the soft palate and uvula, but rarely beyond. The tonsil is deep red or almost purple in colour. Patches of



whitish, dirty yellow, or greyish mucoid secretion are visible, and on their removal superficial bleeding ulcers are exposed. The onset is sudden, but without vomiting. The degree of fever and pulse rate are variable. Erythematous rashes are not uncommon. The tongue is foul, the local pain of definite severity, and the ordinary symptoms of a severe throat affection are present. The throat resembles the condition seen in scarlatina. More or less adenitis is present; temporary and slight, or severe and prolonged and ending in abscess. The course of the illness is slow and there is much disturbance in health. It may end in quinsy.

The *Erysipelatous Inflammation* of the fauces sometimes seen in adults has never come under my notice in children. Local ulceration may start in a crypt. If the mouth of a crypt is blocked, a *Tonsillar Cyst* is formed and projects above the surface as a yellowish-white globular swelling of the size of a pea. It leaves a superficial ulcer on bursting. Tuberculous and syphilitic ulcers are occasionally seen.

*Pneumococcal Angina*.—The pneumococcus is found in a type of membranous sore throat very like that of diphtheria; and also in an acute erythematous type which only differs from erysipelatous inflammation by its slight degree of œdema. Both these forms are rarely fatal but gangrene of the fauces has occurred. As the pneumococcus is found in normal throats it cannot be accepted as the undoubted cause. *Friedländer's Bacillus* has been found by Mayer (1900) in acute and chronic membranous angina.

In streptococcal cases the membrane is thinner, looser, and less adherent than in diphtheria or pneumonia; the temperature more persistent and sloughing more prominent.

*Vincent's Angina* is occasionally seen in children, after 7 years of age, with defective teeth, bad health and unhealthy surroundings. Vincent described a mild type, in which he found fusiform bacilli with staphylococci or streptococci. It is unilateral, superficial and localised. The onset is insidious. There may be glandular enlargement, offensive breath, slight dysphagia and little fever, occasionally up to 103° F. A white, chalky or greyish, soft, false membrane is found on some part of the tonsil, the base of the uvula and edge of the soft palate. It separates slowly by necrosis leaving a shallow ulcer which heals in 7-14 days; the fever only lasts a few days. In a more severe ulcero-membranous type a fusiform bacillus and a spirillum or spirochæte are found. There is greater destruction of tissue, perhaps of the whole tonsil and uvula, with fever and constitutional symptoms, such as scarlatiniform rash, purpura, rheumatic pains, anorexia and constipation; and occasionally albuminuria, myocarditis and endocarditis. Some of these are due to secondary streptococcal infection. The local affection gives rise to foetid breath, salivation, dysphagia, otalgia and submaxillary adenitis. It is usually unilateral, but may attack both tonsils and spread to the soft palate, tongue, gums and



lips. It lasts from 1-3 weeks. The glands rarely suppurate. A certain amount of contraction may follow severe necrosis. Occasionally it is fatal from broncho-pneumonia or toxæmia. The mild cases, in which the necrosed epithelium looks like membrane and hides the ulceration, are liable to be mistaken for diphtheria. But the constitutional symptoms are generally slight. True diphtheria may be a complication or sequel.

*False Membrane on Tonsillotomy Wounds* develops in 24 hours. It is dirty grey, 2 mm. thick, limited to the surface of the wound, and lasts for 5 or 6 days. It consists of leucocytes, fibrin, streptococci, and perhaps staphylococci and pseudo-diphtheria bacilli.

*Quinsy* is sometimes an acute suppurative inflammation of the tonsil, but almost invariably starts in the areolar tissue between the tonsil and the pharyngeal aponeurosis and terminates in resolution or in abscess. Hence, it is variously described as *acute parenchymatous* or *phlegmonous tonsillitis*, or as *tonsillar*, *peritonsillar* or *palatal abscess*. It is comparatively rare in early life. It may be due to infection viâ the tonsil, or secondary to septic or follicular tonsillitis with which it is often associated. It is a unilateral affection, spreading to the soft palate and pillars of the fauces, and sometimes causes a plegmonous inflammation of the pharynx.

The onset is less acute and the local symptoms more severe than in follicular tonsillitis. It is a more local affection with less fever, malaise and muscular pains. There may be a little stiffness, or pain in the throat so severe as to render swallowing impossible. The pain is felt in the neck and, being due to inflammation in the deeper tissues, it may be severe although there is little to be seen in the throat. It may be difficult to open the mouth. The inflammation spreads to the mucosa, and the tonsils, fauces, uvula and soft palate are intensely red and swollen, and often acutely oedematous. The tonsils may be so large as to meet in the middle line, and give evidence of follicular tonsillitis. In a day or two one tonsil and the anterior pillar of the fauces are pushed forward and upward by the swelling, or the swelling may appear on the soft palate in front of the tonsil. The unilateral swelling bulges the palate forward and depresses it, displaces the uvula to one side, and reduces the size of the faucial channel. The tonsil, though enlarged, may be hidden from view. Cervical pain may cause torticollis.

The swelling is more easily recognised by palpation than inspection, and a soft spot, indicating suppuration, more easily localised. In tonsillar abscess the general bulging of the palate is absent, and a yellow area shows that pus is nearing the surface. In peritonsillar inflammation the swelling subsides in 3-5 days, if no pus is formed. Frequently pus may be present in 2-3 days and the abscess bursts spontaneously, through the supratonsillar fossa, above and external to the tonsil and between the faucial pillars, or through the soft palate. If it bursts during sleep the pus may run into the larynx and cause death.



*Gangrene* may be primary or secondary to diphtheria, scarlet fever, cancrum oris and ulcerative affections of the mouth. A purple spot appears and becomes darker in colour. An ulcer forms and necrosis is rapid, with terrible foetor, somewhat faecal in odour. It does not always start in the tonsil or remain limited to it. Death results from sepsis.

*General Diagnosis of Throat Affections.*—The white specks of thrush on the tonsil simulate those of follicular tonsillitis, but there are other patches on the buccal mucosa, tongue, etc. The tonsils are of normal colour, the patches dead white and asymmetrical, and there is neither pain nor adenitis. In measles there is a symmetrical red blush, with tiny red papules, chiefly affecting the palate. Pain is slight or absent, and buccal spots are often present. In scarlet fever there is a symmetrical, vivid red erythema of the fauces, uvula and tonsils, sometimes extending to the roof of the mouth and pharynx. The buccal mucosa is unaffected and there is no early adenitis. Pain may be considerable and a strawberry tongue present. Later on there may be yellowish-white or dirty white deposit, soft and easily broken up, with ulceration. In diphtheria the child is peculiarly placid in aspect. The tonsil is a little reddened, and there is a unilateral, tough, adherent deposit or membrane with definite edges, not limited to the tonsil, and definite odour. Early adenitis, little or no pain, and laryngitis are often present.

*Treatment of Throat Affections.*—The general measures consist in bed and a liberal diet of soft and nutritious food. An initial purge is necessary in acute attacks. Quinine, salol or salicylates may abort attacks, if given freely, but salicylates are of no use in diphtheria and scarlet fever. Phenacetin and phenazone relieve headache and restlessness. Febrifuges are useful; alcohol, if there is cardiac weakness; iron, nux vomica and strychnia, as tonics. It may be necessary to paint with cocaine 1-5 per cent., to relieve pain and permit deglutition. Local measures include hot gargles, fomentations and poultices; ice to suck and an ice-bag externally; and rarely leeches. Douching, irrigation and gargling are rarely suitable for children. Such solutions as sanitas 1 per cent., liq. calc. chlorinatæ 0·5 per cent., liq. sod. chlorinatæ, chlorine water and very weak sulphurous acid can be used; or various combinations of the chloride, bicarbonate, biborate, salicylate and sulphate of soda and boric acid p.a., 1-3 drs., with 2 drs. of cherry laurel water, to the pint of water may be used. A syphon douche should consist of a vulcanite end-piece attached to a long rubber tube, and be inserted between the back teeth. The child must be firmly wrapped up in a sheet, the arms confined, in a sitting posture and head bent forward, with the nurse's left arm round the head and pressing it to her side. Sprays of sublamin, a non-toxic mercurial, 1 in 1,000, izal or cyllin 1 in 1,000, perchloride or biniodide of mercury 1 in 2,000, glycothymolin 1 in 4, or liq. sod. chlorinatæ 1 in 8, can be used. On the whole, if local applications are necessary, it is



best to paint or swab with pure glycothymolin, glycer. ac. tannici, ichthyol and water p.a.; tr. benzoin. co. and glyc. p.a.; liq. sod. chlor.; resorcin grs. 10 to 1 oz.; or liq. hyd. perchlor. and glyc. p.a. Formamint lozenges should be sucked frequently. The insufflation of powders is unsatisfactory. In young children the struggling and distress involved in local treatment may be more injurious to the strength of the child than is compensated for by the benefit derived from it.

Surgical measures are required in quinsy, if there is much swelling, œdema or any sign of laryngeal obstruction. Even if pus is not found, local bleeding does good. No anæsthetic is permissible for there may be glottic œdema. Cleanse the throat and apply a 5 per cent. cocaine spray. With the child in a sitting posture, the head steadied and the throat well illuminated, push a pair of Lister's sinus forceps into the soft or boggy spot, backwards and a trifle outwards, and open them as they are taken out. The soft spot is generally situated in the soft palate, just external to the supratonsillar fossa, at the point where a vertical line corresponding to the anterior faucial pillar crosses a line drawn horizontally across the base of the uvula. If a scalpel is used, it must be protected by sticking plaster up to the last  $\frac{1}{4}$  in. It is inserted in the same situation and a cut made upwards and inwards.

*Enlarged Tonsils.*—Enlargement of the tonsils is etiologically due to the same causes as adenoids. It takes place in a vertical, antero-posterior or horizontal direction. The surface is normal, rough and irregular, patchy or honey-combed with irregular holes, and occasionally papillated. There are two types, the hard and the soft, depending upon the relative amount of lymphoid and fibrous tissue. The crypts are dilated and filled with cheesy matter in the soft variety, or obliterated by the contraction of the excessive fibrosis in the hard variety. Usually the two conditions are more or less combined.

Chronic parenchymatous tonsillitis is common in children and often associated with adenoids. The tonsil is hard, round and smooth. Chronic lacunar or follicular tonsillitis is liable to cause toxæmia and fever from absorption of decomposing matter in the crypts. The tonsil is large, soft, and irregularly pitted.

The *symptoms* ascribed to enlarged tonsils are commonly due to adenoids, so tonsillotomy alone may prove a failure. Enlargement causes difficulty in swallowing; interference with mouth breathing, a serious trouble if nasal respiration is obstructed by adenoids; a thick, indistinct voice; deafness from upward pressure of the soft palate and imperfect middle ear aëration; and maldevelopment. The lungs are imperfectly expanded, the ribs sink in and the child may become pigeon-breasted. The blood is imperfectly aërated and general health impaired. Enuresis and foetal incontinence occasionally occur. The local conditions and the open mouth lead to congestion, the entrance and growth of microbes, attacks of



tonsillitis, and enlargement of the upper set of the deep cervical glands, which may suppurate. Reflex cough is produced. It is dry or hacking and almost constant, or may be paroxysmal and spasmodic. The local irritation causes discomfort in the throat and perhaps pain shooting to the ears. Toxæmia and adenitis are the most important complications.

If the tonsils are small, hard and round, and contain no open crypts, local treatment may be sufficient and spontaneous atrophy may occur at puberty. The soft tonsils may diminish in size after acute inflammation, or remain stationary after attaining a certain size, until they decrease at puberty. As a rule they improve little during childhood and they increase the liability of the child to acute inflammation, septic infection, scarlet fever, diphtheria, etc. They exaggerate the gravity of these diseases when they occur and increase the liability to ear disease.

Local treatment consists in the application of astringents for the soft variety, and compound tincture of iodine in either type. Cod-liver oil, iodide of iron, sea air and a high dry climate are beneficial. Provided hypertrophy is not due to recent tonsillitis, operation should be done if there are distinct mechanical obstruction, recurrent attacks of catarrh, reflex cough, ear trouble or adenitis, and if the crypts remain blocked with caseous matter or discharge foetid, purulent or muco-purulent secretion. The operation should depend upon the symptoms and not on the age or the size, unless they are very large. It is not urgent in mild cases at about puberty, unless singing is taught.

*Tonsillotomy.*—An anæsthetic is required for a nervous child or if adenoids are removed as well. A recumbent position and a gag are then necessary. In other cases 5-10 per cent. solution of eucaine is painted on, and the tonsils are removed by a spade-shaped guillotine with the child in a sitting posture. The instrument should be warmed, and care taken to first encircle the lower edge of the tonsil and to press firmly outwards. Adhesions of the pillars of the fauces, if present, must be separated first. The operation is often imperfectly done, a small slice only being cut off. The portions left are liable to recurrent inflammation. The small pad left is said to protect the carotid artery, and to prevent an excavation in which food may collect and decompose. The dangers of the operation are slight. With ordinary care the carotid cannot be wounded by the guillotine or a blunt pointed bistoury. An abnormal vessel may be opened. Usually the patient hardly suffers and bleeding stops in a few minutes, especially if the patient stands up after the operation. Enucleation is a more complete cure and must be done under anæsthesia. The tip of the finger is inserted into the supratonsillar fossa and the gland separated from above downwards. It is then seized with tenaculum forceps, pulled inwards and rotated, thus twisting and tearing the vessels, and twisted or cut off. There is little bleeding, no late hæmorrhage, and no possibility of recurrence. It is easy in the fibrous form, but in the soft variety is



more difficult. The raw surface soon heals. Discomfort and pain may last two days.

Immediate hæmorrhage after tonsillotomy is rarely serious, but recurrent hæmorrhage is dangerous and liable to be overlooked for the blood is swallowed. For general oozing give ice to suck, paint the surface with adrenalin solution, or apply digital pressure, styptic cotton, or a mixture of tannic acid 3 parts and gallic acid 1 part, in sufficient water to make a hard mass, rubbing it in freely and applying external pressure at the same time (Hovell). If there is a single bleeding point, the severed tonsillar artery, the vessel should be clamped with a pair of Spencer-Wells forceps left in situ. Ligature of the carotid is probably never required. Bleeding is venous or capillary, and ceases as soon as faintness supervenes. It may be kept up by measures adopted for its prevention. Another method is to apply gauze and stitch the pillars together for 24 hours. Secondary hæmorrhage may result, from the separation of a slough opening up a small vessel, as late as the sixth day. Sepsis and septic pneumonia are possible sequels and septic throat affections sometimes ensue. Caries of the teeth should be attended to before operation, and antiseptic precautions adopted. Although many cases are operated on in hospital out-patient departments with impunity, it is preferable that the operation should be performed at home or in hospital, and the child put straight to bed in a warm room and kept there for 2 or 3 days. Exposure to cold may set up inflammation, and exercise may bring on recurrent hæmorrhage. The child should be fed on boiled milk, cold or lukewarm, and then soft food until able to swallow comfortably. After food the throat should be washed out or sprayed with an alkaline antiseptic lotion. The wound looks grey on the day after operation (p. 233). Sepsis is indicated by an ashy grey slough, furred tongue, offensive breath, adenitis, painful deglutition and fever. It is treated by calomel, quinine, perchloride of iron, and chlorine spray or gargle.

*Tuberculosis.*—Both the faucial and pharyngeal tonsils may be infected by food, inhalation, through the blood stream, or by coughed up sputum. Probably the infection is mainly by the blood stream. Possibly the bacillus can pass into the lymphatics and infect the glands without causing a primary lesion of the tonsils. If the tonsils are tuberculous, the follicular glands at the base of the tongue and the cervical glands are likely to be infected. Before asserting that the tonsillar affection is primary the relative age of other tuberculous foci, if present, must be considered.

Friedmann found evidence of tubercle in 17 out of 145 tonsils removed at operation or after death. Latham, by inoculation experiments, demonstrated the presence of the bacillus in 7 out of 45 similar cases, the youngest being 3 months of age. Sukchito Ito (1903) only found 6 cases of infection in 104 children under 13 years of age; none primarily tuberculous. He examined 8,000 sections.



Considering the frequent exposure of the pharynx to infection, primary tuberculosis of the pharyngeal tonsil is very rare. It may lead to cervical adenitis. A few cases have been described in children. Inoculation experiments are unreliable for surface contaminations cannot be excluded, and tubercle bacilli are sometimes found in the healthy.

**Pharyngeal Affections.**—Periodical attacks of congestion in the pharynx are associated with nasal catarrh or may start in the nasopharynx. They are described under the name of *Acute Catarrhal Pharyngitis*, *Pharyngitis Superior*, *Adenoiditis*, *Pharyngeal or Retro-nasal Angina*, and *Post-nasal Catarrh*. The symptoms are fever, slight nasal irritation and snuffles, nasal voice, muco-purulent discharge, earache, deafness; cough, especially during sleep; paroxysms of dyspnoea, occasionally at night; and interference with swallowing. Tenacious mucus is visible on the reddened and swollen posterior pharyngeal wall. The attack lasts from a few days to a week or more, and may end in secondary bronchitis. It produces adenoids or chronic hyperplasia. The local treatment is that suitable for adenoids.

*Follicular or lacunar pharyngitis* causes fever and general malaise. It is often overlooked, for it may be only diagnosable by posterior rhinoscopy. The pharyngeal tonsil is covered with patches of follicular secretion, similar to those seen in follicular tonsillitis. The follicles may open into a small recess in the middle line with a single orifice. The exudation may be excessive and coherent into a membranous structure simulating diphtheria. It may extend to the Eustachian tube and cause earache, otitis media and even meningitis. Occasional complications are parenchymatous tonsillitis, quinsy, adenitis, erythematous rashes, and laryngeal or respiratory catarrh. It may simulate typhoid fever because of fever, gastric symptoms and enlarged spleen; or meningitis, if the nervous symptoms are pronounced. The treatment consists in local applications to the pharynx, by a large suitably curved brush, and nasal douching.

*Pneumococcal Pharyngitis* is generally associated with tonsillar inflammation. Elliott, of Chester (1909), has drawn attention to cases of this type in children and adults. They were characterised by pharyngitis, a temperature up to 102-103° F., sudden onset, and rapid cervical adenitis of the submaxillary, deep cervical glands, and those below the lobule of the ear. The duration was only a few days, and there was no ulceration. In some respects this is like glandular fever (q.v.).

*Pharyngeal or Post-adenoid Abscess* is due to secondary infection, via adenoids, and is situated in the connective tissue between the pharyngeal tonsil and aponeurosis. The symptoms are those of adenoids, combined with recent sore throat and rapid increase of nasal obstruction. There is no difficulty in swallowing unless the pharyngeal catarrh is general. A cystic swelling, usually median and perhaps extending to the roof of the naso-pharynx, is felt, on digital examination, covered by adenoids. It is



evacuated by the knife or by pushing sinus forceps into the centre of the swelling.

**Adenoids.**—*Syn. : Post-nasal growths or adenoids—Adenoid growths—Adenoid vegetations in the naso-pharynx.*—The name of adenoids should be limited to hypertrophy of Luschka's gland, a circumscribed mass of lymphoid tissue in the upper part of the posterior wall of the naso-pharynx in the middle line, oval in shape with the long axis vertical. It is roughly divided into two lobes by a median longitudinal groove and is covered by columnar ciliated epithelium. This gland is present at birth, varies in size, at its maximum between 4-6 years of age, and then slowly atrophies. It is impossible to draw a sharp line of demarcation between a physiological and pathological hyperæmia and hyperplasia of the gland. Its removal may be insufficient for cure, because the rest of the adenoid tissue of Waldeyer's ring may be hypertrophied, perhaps to even a greater extent. Large masses may be situated round the posterior choanæ and Eustachian tubes. This is flatly contradicted by some surgeons who state that this region may be safely left alone.

*Etiology and Pathology.*—Heredity is a potent factor for a tendency to lymphatic hypertrophy or hyperplasia is present in many children of the same family. Adenoids are constantly associated with enlarged tonsils. Important factors are tuberculosis, infective fevers, liability to catarrh of the respiratory passages, bad hygienic surroundings, climatic conditions, and neglected colds. Some of these tend to produce lymphatic hyperplasia and others to cause catarrhal affections. Interference with normal breathing through the nose may be a result and not a cause. Nearly all infants with adenoids are rachitic, but rachitic children are constantly free from adenoids. A highly arched palate is liable to occur in rickets, catarrhal affections are common, and the small posterior nares are easily blocked by the swollen lymphoid tissue. The increase in size from a small patch to a mass filling the naso-pharynx is due to inflammation or hypertrophy, and the growth is soft or hard according to the degree of fibrosis. It may be quite hard and fibrous. Occasionally cheesy nodules can be squeezed out. The central recess may be partly converted by adhesions into a cavity (*Tornwaldt's Bursa*). The adenoid tissue may give rise to trouble from the moment of birth, such as difficulty in breathing, stridor and inability to suckle. Operative treatment is not often necessary before 4 years of age.

The *Symptoms* are local and constitutional; nasal, respiratory and affecting deglutition; obstructive, catarrhal and reflex. The local symptoms indicate the degree of obstruction. The child is a mouth-breather and has a stupid appearance because of the half-opened mouth. Hence arise dryness of the mouth and throat, difficulty in swallowing, inability to suck freely without stopping to take breath, noisy breathing and snoring during sleep, perhaps amounting to a congenital stridor in infants or attacks like laryngospasm, early decay of the teeth, and



imperfect oxygenation of the blood. To the indirect effects of imperfect respiration and mouth-breathing are ascribed the narrow, highly arched palate, long and narrow face, narrow nasal fossæ, deformities of the upper jaw and of the chest. Recession of the lower ribs is common in infants. Head sweating and "snatchy" sleep are due to deficient oxygenation. The voice is stuffy and dead. There is difficulty in pronouncing certain consonants, M and N pronounced as EB and ED, backwardness in learning to articulate clearly and perhaps stammering. Speech is most likely to be defective in the congenital cases. The nasal signs include lateral compression of the anterior nares, snuffling, recurrent or chronic mucoid, muco-purulent or purulent discharge, periodical "colds," sometimes epistaxis, and occasionally paroxysmal sneezing attacks like those of hay-fever. Respiratory catarrh, hoarseness and asthmatical symptoms may occur. Cough is variable, short, dry, hacking, barking or choking in character; the most typical type is a paroxysmal cough which may simulate whooping cough. It may be constant while awake; occur chiefly on lying down; wake the child up during sleep, or be worst in the early morning. Its persistence may render operation necessary, and its severity may cause vomiting or even involuntary defæcation. Ear troubles are very important. At first there may be slight attacks of earache, and then recurrent and more severe ones; a gradually increasing or recurrent deafness; and finally attacks of otitis media and otorrhœa. The tympanic membrane is depressed. The cervical glands are often enlarged and liable to septic infection or to become tuberculous. The vacant, semi-idiotic facial aspect, due to the half-open mouth, gives the impression of mental dulness or hebetude, which is more apt to be due to deficient aëration. The child may be backward, but the intellect unimpaired. Deafness increases the appearance of stupidity and causes backwardness, inattention and listlessness. Moral degeneracy has improved after operation. The general health and nutrition are impaired because of imperfect expansion of the chest and deficient oxygenation. Malnutrition may be due to the difficulty of sucking. The child cannot breathe and suckle simultaneously.

The chief indirect effects arise from imperfect aëration of the blood or reflex irritation, and include enuresis nocturna, night terrors, moaning, teeth-grinding, somnambulism, frontal headache; torticollis, secondary to adenitis; rarely, a staggering ataxic gait and recurrent vomiting. Many of these are merely coincident. Laryngeal complications include "congenital crowing" and laryngospasm. Some of the nervous phenomena are due to the catarrh and can be cured by treatment of the catarrh. Breathing may be noisy from birth and at times distressed, and the crowing very loud. It persists day and night, and must be distinguished from true congenital stridor.

*Diagnosis.*—In infancy the symptoms of adenoids have to be distinguished from those due to other causes, notably simple post-natal



catarrh and the snuffles of congenital syphilis. The stridulous breathing differs from congenital laryngeal stridor in that it is most marked during sleep, when the mouth is shut, and while suckling. At all ages the presence of adenoids is diagnosed by the interference with breathing and the functions of the nose and naso-pharynx, and the presence of enlarged tonsils. The "*Adenoid Facies*" is merely the appearance due to nasal obstruction from any cause.

If necessary, the naso-pharynx must be examined digitally. This is difficult in infants because of the small size, and in older children it is unpleasant and alarming, if they are nervous. The examiner stands on the right side, somewhat behind the child, places the left arm round the head, and depresses the lower jaw with the left hand or pushes the left cheek between the teeth. The index or little finger is then inserted along the right faucial pillar and up into the naso-pharynx. This mode of examination is not of very much value, for the diagnosis is based mainly on the symptoms and the advisability of operation on the severity of the symptoms. It is necessary sometimes, or cases will be operated on although no adenoids are present. Posterior rhinoscopy is a better method of examination and is possible after the third or fourth year, if the tonsils are not too large.

Attention must be directed particularly to mouth-breathing, snoring, nasal obstruction even if only nocturnal, earache, recurrent deafness, otitis, otorrhœa and adenitis, together with the effects on the general health and development of the child, especially of the thorax. Nasal obstruction and deafness due to other causes must be excluded. Care must be taken not to ascribe backwardness, physical or mental, to adenoids, unless there is deafness or very definite obstruction.

*Prognosis.*—Adenoids tend to atrophy at puberty, and the degree of obstruction is reduced by the rapid enlargement of the throat. If they are left alone in early life, they are liable to cause ear troubles and general maldevelopment of the nose, naso-pharynx, upper jaw, palate and chest. The mouth is kept open, the upper lip raised, the incisor teeth become prominent, the teeth are more liable to caries, the voice is nasal, the chest pigeon-breasted, growth stunted, and lateral curvature not uncommon.

The prognosis of operation depends upon the age of the patient and the duration and degree of the obstruction before operation; on the complete removal; on the presence of other defects; and on the removal of the posterior ends of inferior turbinals, if they are hypertrophied. If there is any anterior nasal obstruction, it must be treated first, otherwise mouth-breathing is certain to persist after operation. In many cases the operation does not prevent mouth-breathing, for it has become a habit and may be due to other causes; nor does it always prevent deformity of the jaw and ear troubles. Too much is usually expected. The best results are obtained in those cases in which the obstruction is definitely interfering



with aëration of the blood, expansion of the chest, and the general health.

*Treatment.*—Careful attention to catarrhal conditions of the nose and naso-pharynx prevents the occurrence of adenoids. The general treatment consists in hygiene, sea air or a dry equable climate, teaching the child to breathe deeply and freely through the nose, suitable diet, and tonics such as cod-liver oil, arsenic and iron. Lapayer (1901) recommended the administration of tr. iodi. co., m. 5-30, t.d.s. This is an alcoholic solution of iodine, and is said to cause atrophy of the vegetations. An alkaline nasal lotion removes mucus and relieves the symptoms. Local applications are useful when the adenoid tissue is soft and gelatinous from temporary congestion. The naso-pharynx may be painted with equal parts of liq. ferri perchlor. and glycerine, twice a week, using a laryngeal brush on a curved handle, and then once a week; or with iodine grs. 2, pot. iod. grs. 30, glycerine oz. 1; or alum drs. 2, tannin dr.  $\frac{1}{2}$ , glycer. oz.  $\frac{1}{2}$ , aqua rosarum ad. oz. 2. Sprays are of comparatively little value. If the mass is hard and fibrous it must be removed.

Breathing exercises, so often recommended, are frequently injurious. It is by no means uncommon to find an unfortunate child doing breathing exercises two or three times a day, although on every inspiration the ribs sink in and the sternum is protruded; or perhaps the lower part of the sternum and the cartilages are sucked in. They are only justifiable and useful when the chest is fully and normally expanded on deep inspiration. The child stands in the erect posture and slowly elevates the extended arms until the hands meet above the head, taking at the same time a deep nasal inspiration with the mouth closed. This is repeated 8-10 times a minute for 15 minutes, twice a day. Breathing exercises may improve breathing, but they will not cure hypertrophy of adenoid tissue.

*Operation.*—There are several objections to the unhesitating recommendation of operative treatment. It is an operation which causes anxiety, trouble and expense. Death may result from the anæsthetic or hæmorrhage, immediate, recurrent or secondary. Post-operative hæmorrhage is generally tonsillar. It may depend on hæmophilia. Portions of growth may get into the air passages and cause suffocation or septic pneumonia. Acute otitis media and septic meningitis are occasional sequels. The enlarged cervical glands may become infected and suppurate. Septic infection of the pharynx may occur.

Age is an important consideration. Infancy is no bar. If puberty is approaching the operation may sometimes be postponed. Recurrence is more likely in infants, but this is no objection if the operation is necessary on account of interference with suckling or otitis. It is practically devoid of danger, and adenoids produce the greatest evil effects in infancy. If there is any nasal obstruction, the operation should be done before the commencement of the second dentition. Catarrhal swelling, due to colds



or fevers, may subside after a few weeks or months of local and general treatment.

The chief indications for operation are the duration, persistence and severity of the effects, notably habitual mouth-breathing; imperfect development or deformity of the chest; deafness, depressed tympanum, earache and otitis; constant or recurrent nasal discharge and nasal obstruction; irritating cough, suffocative attacks, bronchitis and asthmatic symptoms; catarrhal affections of the fauces, and adenitis. In older children it is advisable in order to improve the singing voice.

Recurrence is rare, if the operation is thoroughly done after the fourth year. If relief is not obtained the nose must be examined for deflected septum, hypertrophied turbinals, mucous polypi and anterior nasal stenosis. After operation there is no immediate recovery, for the habit of mouth-breathing and the speech defects are only slowly cured, and may persist throughout life if operation has been unduly postponed. General treatment and breathing exercises are useful at this time.

The operation is done under anæsthesia, A.C.E. or  $C_1E_2$  mixture, but not nitrous oxide gas, with the child in the dorsal position on a flat table. The tonsils may be removed afterwards, unless so large as to interfere with operation; and the posterior ends of the inferior turbinals are removed, if they are hypertrophied. Kirstein's modification of Gottstein's curette and St. Clair Thomson's adenoid curette are the best instruments, and generally preferable to the finger nail, the steel nail or forceps. Subsequent to operation swallowed blood may be vomited; and snoring may persist and be worse for 2 or 3 nights, or until blood and stagnant discharges are cleared away. The child is kept in bed 2-4 days, given an aperient on the second day, soft food for 3 days, and light diet for a week. An antiseptic alkaline lotion is used for nasal stuffiness and offensive breath, as a spray or mild douche.

If there is glandular enlargement, it is advisable to put the child on a course of arsenic before operation. The arsenic seems to render the glands less susceptible to septic infection and suppuration, an accident which sometimes follows operation. Occasionally a nasal tone after operation is due to paresis of the soft palate, and is cured by cold water gargles.

**Retro-Pharyngeal and Retro-Œsophageal Abscess.**—The true retro-pharyngeal abscess is situated between the bucco-pharyngeal aponeurosis and the prevertebral fascia. The tuberculous abscess of cervical caries is posterior to the prevertebral fascia. It begins insidiously in the middle line and spreads slowly outwards. The post-adenoid abscess (p. 238) is in the middle line anterior to the bucco-pharyngeal aponeurosis. The ordinary retro-pharyngeal abscess is due to infection of the glands in the pharyngeal connective tissue, from septic conditions of the naso-pharynx and tonsils, injury by fish bones, and specific fevers, such as measles and scarlet fever. It is an intra-pharyngeal abscess starting behind the posterior



pillar of the fauces, spreading backwards to the posterior wall and then inwards. In its early stage the small rounded swelling can be felt and often seen behind the tonsil, and the child is ill. As the swelling enlarges, becoming tense and elastic, it give rise to salivation, dysphagia and muffled cry; and the head is thrown back or to one side. Then the signs of obstructive dyspnœa develop, but stridor and croup are generally absent.

The onset is gradual, with catarrh of the nose and throat and often a purulent rhinitis. The child is generally brought for anorexia or dysphagia, vomiting, cough, croup or bronchitis. The tongue is furred, the fauces red and covered with mucus, and the tonsils enlarged, one perhaps pushed forward. The head is retracted and the neck stiff or rigid, and looks swollen because of unilateral or bilateral secondary adenitis. Dyspnœa is inspiratory at first and later expiratory, with recession. It may be paroxysmal. The cry and voice are altered, hoarse or crowing, cough spasmodic, and snoring present when the child is in the prone position. The dysphagia varies with the size and position of the abscess. Sometimes food regurgitates through the nose. The child looks ill, is restless, sweating, pale or cyanosed, with the mouth open, rapid pulse, and temperature up to 104° F. It refuses food because of anorexia or difficulty in swallowing.

If the abscess is in the upper part of the pharynx, the signs are mainly an open mouth, snoring, snuffling, nasal cry and interference with nasal breathing. If the abscess is low down, the cry is laryngeal, breathing stridulous, the respiratory murmur diminished, and there are many moist sounds.

These cases generally occur between 6 and 12 months of age and are rare after the third year. Bókay's figures are 296 in the first year, 78 in the second, and 93 afterwards. They are extremely fatal if untreated, death resulting from asthenia, asphyxia, glottic spasm or vagus inhibition. Rupture is rare, and may cause suffocation or inspiratory pneumonia. Ulceration has taken place into the carotid artery and into the middle ear or external auditory meatus. About 5 per cent. of those treated by incision do not recover.

*Diagnosis.*—Extra-pharyngeal abscess is due to suppuration in deep cervical glands and preceded by glandular enlargement in the neck. There is more external swelling. The abscess bulges but rarely bursts into the lateral wall of the pharynx. It seldom causes serious obstruction to breathing or swallowing. Large mucous cysts and a retro-pharyngeal blood cyst, in purpura, cause symptoms like those of retro-pharyngeal abscess. Cases have also to be diagnosed from croup, œdema of the glottis, laryngeal obstruction, foreign bodies, enlarged tonsils and adenoids, diphtheritic palsy, and pharyngitis or rhinitis, if high up. The rigidity of cervical caries is distinguished by its long duration; that of simple abscess comes on suddenly and is often absent. Palpation is more reliable than inspection. The pharynx is explored from above downwards and from side to side.



A gag must not be used, as it may cause rupture of the abscess. The swelling is lateral, larger, and usually lower than that of post-adenoid abscess.

*Treatment.*—The preventive treatment is that of tonsillar and nasopharyngeal affections. The abscess must be opened through the mouth, and the child turned on its face as soon as the incision is made. Use a straight scalpel,  $\frac{1}{3}$  in. long, and no anæsthetic; open the mouth with the finger or tongue depressor, but not very wide. If the erect position is chosen, bend the child forward immediately after the incision is made. Sudden respiratory failure may arise from manipulation of the pharynx or the use of a tongue depressor. Other accidents at operation are sudden syncope, spasm of the glottis, hæmorrhage from a branch of the carotid, and entrance of pus into the air passages. Secondary infection is rare. The abscess may require re-opening and expression. An extra-pharyngeal abscess and that due to cervical caries are opened by incisions through the neck, generally along the posterior border of the sterno-mastoid.

*Retro-Œsophageal Abscess* is due to spinal caries, diphtheritic pharyngitis, a caseous mediastinal gland, or extension of suppuration from other parts. The correct diagnosis may be impossible. Dyspnœa is present to a certain extent in all cases, and in many is a prominent and urgent symptom, with recession. The voice is little or not at all affected. Cough is present, perhaps for some months, and may be croup-like or metallic, suggesting laryngeal mischief, stenosis or pressure lower down. Unless the abscess is partly retro-pharyngeal dysphagia is absent, for the œsophagus is easily pushed to one side. It may occur temporarily from rupture into the œsophagus. There may be swelling in the neck on both sides, tenderness, pain on movement and fever. Some of these abscesses burst into the œsophagus or burrow into the tissues of the neck. Rupture into the œsophagus may be followed by secondary stenosis or a diverticulum. The abscess must be opened at the posterior edge of the sterno-mastoid, and the child fed nasally, if unable to swallow.

**The Œsophagus.**—*Congenital Malformations*—*Inflammation*—*Ulcer*—*Foreign Bodies.*—The œsophagus appears in the fœtus at the end of the fifth week as a cylindrical tube. In birds, fishes and reptiles it persists as an impervious cord for a long time. Complete absence has been recorded in 5 cases up to 1902. More commonly it is obliterated or partially stenosed. Other malformations are, a double tube with reunion at the lower end—very rare; diverticula or pouches, situated in the middle line and not causing symptoms till late in life, but possibly of primary congenital origin; fistulous communication with the trachea or bronchus, generally associated with atresia; fistula of the neck, due to imperfect closure of the second and third branchial arches involving the œsophagus; the lower end opening into the trachea just above the bifurcation, while the upper one ends in a dilated cul-de-sac.



*Atresia or Occlusion* is more frequent than supposed. Hirschsprung collected 14 cases, 11 verified post mortem, of which no less than 7 occurred in the Copenhagen Lying-in Hospital during 16 years. Thomas (1904) added 6 more; and Villemin (1904) another one. The occlusion is situated at the junction of the œsophagus with the pharynx, or in any portion of the tube. In the most common variety the upper part ends in a dilated blind pouch and the lower one opens into the bronchus or trachea, generally just above the bifurcation or below the larynx. In the less common type the segments are united by a fibrous or muscular cord  $\frac{1}{2}$ -1 in. long. It is due to maldevelopment of the tracheo-œsophageal septum, and is often associated with other defects of the gut.

The child regurgitates all its food through the mouth and nose, and has severe choking fits with cyanosis from entrance of food into the larynx. It is hungry, micturition scanty, the bowels cease to act, and it dies from inanition in a few days. Life may be prolonged by rectal feeding and gastrostomy. The diagnosis is confirmed by the finger in the pharynx or by passing a bougie.

*Stenosis or Stricture* is congenital or acquired. Diffuse dilatation is rare and may be due to muscular atony and cardiospasm. A *congenital stricture* is commonly situated at or below the bifurcation of the trachea. It varies in length, usually about  $\frac{1}{2}$ -1 $\frac{1}{2}$  ins., and is fibrous in structure. Above the stricture the tube is more or less dilated. More rarely a partial stenosis is due to a fold of mucous membrane, like a diaphragm, at the upper or lower end. The minimum diameter of the œsophagus in the newborn is 4 mm., and the distance from the gums to the cardiac end of the stomach is 17 cm. A soft catheter is passed for diagnosis. It may go into the trachea. Symptoms rarely develop until 6-18 months after birth, and consist of regurgitation of food, without nausea and often mistaken for vomiting. Food is generally retained unless regurgitated at once; that brought back has an alkaline reaction, unless acid to start with. The appetite is good, but the child becomes pale and emaciated.

*Acquired stricture* is most common at 3-9 years of age, due to swallowing caustic potash (lye). In Norway and Sweden, where lye is used instead of soap for cleaning purposes, it is drunk by children in mistake for milk. The burns on the lips, tongue and pharynx may be slight and even absent, though marked in the middle or lower end of the œsophagus. The stricture is ring-like or tube-like, and usually in the upper third. It may be complete and cause rapid inanition; or incomplete and intermittent, partly due to spasm on deglutition. Symptoms come on after 2 or 3 weeks. A bougie may be used 3 or 4 weeks after the injury, and then 3 times weekly for 6 months. The general treatment of stricture is dilatation by bougies, preceded by gastrostomy and dilatation from below upward, if the stricture is complete and the tube dilated above.

*Œsophagitis* may be caused by infectious disease, but is more commonly due to caustics, acids and hot water. Diphtheria causes longitudinal



erosions with sharp cut edges covered with membrane, or the whole tube is covered. In scarlet fever the inflammation is diffuse and secondary ulceration ragged. It is due to extension from the throat and may end in stenosis. Slight burns produce superficial necrosis. Severe ones cause extensive destruction, ulceration, cicatrisation and stricture. Bloody mucus and even portions of mucous membrane are brought up. Lye is fatal in 25 per cent., sulphuric acid in 50 per cent. (v. Hacker). Serious stricture follows in 50 per cent., of which one-third are fatal. Sloughing, hæmorrhage, perforation, cellulitis, mediastinitis, pyopneumothorax and emphysema of the skin may occur.

*Ulcer* may be due to perforation by a caseous gland or to a broken-down pock in variola. More extensive ulceration follows on inflammation. Simple ulcer is rare, and is probably a peptic ulcer dependent on frequent vomiting. A few cases are on record in which a simple ulcer has been found just above the cardia, or at its junction with the Œsophagus, in the first few days of life ; death being due to hæmatemesis or melæna.

*Foreign Bodies* are often passed, per anum, without trouble after a period of time varying with the nature of the object and the age of the patient. Sometimes they stick in the pharynx, or more often just behind the level of the upper notch of the sternum. Occasionally they pass through the stomach or intestine into the subcutaneous tissue of the abdominal wall, and even to more distant parts. Sometimes they are wilfully introduced, e.g., corks. The most common foreign body is a coin which has been put in the mouth and accidentally swallowed. Coins lodge at the commencement of the Œsophagus, just above the cardia or, most frequently, opposite the bifurcation of the trachea at the level of the upper limit of the sternum and the junction of the seventh cervical and first dorsal vertebræ. Here there is a slight fusiform dilatation, due to constriction by the transverse aorta passing just below. The coin lies against the anterior wall and fluid food can pass behind. The distance from the upper incisors in infants to the lower end of the coin is 15 cm. It may give rise to brassy cough, regurgitation of food, inability to swallow solids, fever and symptoms of ulceration, such as foul smelling material brought up at intervals. Although coins have been retained for months and even years, and then vomited or coughed up, the dangers of ulceration are so grave that they should be removed at once. The child is examined and the coin removed while in a sitting posture in front of the X-ray screen, or anæsthetised and horizontal. Bougies, probangs and coin catchers are dangerous in cases of long standing, and in recent ones may push the coin further down. Forceps or Kirmesson's single hook should be used ; sometimes Œsophagotomy is necessary. A recently swallowed foreign body may sometimes be dislodged by inducing forcible retching with a tongue depressor or by emetics. Such treatment is dangerous if the body is rough or pointed. Substances, such as a bolus of unmasticated food, stuck in the fauces can be dislodged by the finger,



## CHAPTER XXII.

### THE STOMACH.

*Malformations — Dilatation — Gastric Contents — Signs of Gastric Disease*  
--- *The Appetite — Pica— Vomiting — Regurgitation — Rumination—*  
*Functional and Inflammatory Disorders—Gastric and Duodenal Ulcer.*

At birth the stomach is almost cylindrical and is placed obliquely in the epigastrium. The cardiac end is just to the left of the tenth dorsal vertebra. The pylorus is in the middle line and is the lowest segment when the stomach is empty. The fundus is relatively flat, gradually develops and becomes more and more transverse. The gastric capacity is discussed in the chapter on diet (p. 52). *Malformations* are rare. Sometimes the stomach is situated in the thorax as part of a diaphragmatic hernia. *Hour-glass contraction* is a pathological curiosity, probably secondary to ulceration. It is usually in the middle third, less often near the pylorus. It has not been found in the foetus. *Post mortem constriction* must not be mistaken for true hour-glass contraction. *Diverticula* have been found arising from or near the pyloric canal, and containing pancreatic tissue. Accessory pancreatic tissue is not uncommon in the walls of the stomach and duodenum.

**Acute Dilatation** is rare. It may occur from no apparent cause. A healthy breast-fed infant, 9 months old, was found dead in bed two hours after being suckled. The stomach was enormously distended with gas and contained a small quantity of clotted milk (Belilos, 1903). A healthy bottle-fed infant, 11 months old, was laughing and playing on its mother's knee shortly after being fed, when it seemed to stop breathing, "made a noise in its throat," and died. The stomach contained 19 oz. of fairly thick Mellin's food and gas (P. R. Cooper, 1903). Similar dilatation may occur in acute diseases, e.g., scarlatina, typhoid, pneumonia and pericarditis; and less often in chronic illness. It is a rare sequel after chloroform anæsthesia, probably due to paralysis of the solar plexus. This variety causes enormous distension, excessive green vomiting and constipation. Sometimes the pylorus and duodenum are also widely dilated. It is treated by lavage, strychnia sub cutem, and no fluid by mouth.

**Chronic Dilatation** in a mild form is almost invariable in the bottle-fed, from excess of food. Rickets is an important cause. The unsuitable food which has caused the disease is liable to undergo fermentation and



set up catarrh, and the muscle wall is atonic. Dilatation is common in all pulmonary affections in infants and may prove fatal by upward pressure on the diaphragm. It is a characteristic feature in pyloric obstruction (q.v.). Symptoms may be limited to those of chronic dyspepsia, unless there is pyloric obstruction, when characteristic vomiting and visible peristalsis ensue. The outline of the greater curvature is easily seen. The treatment is that of the cause, *plus* lavage and strychnia.

**Gastric Contents.**—The reaction is neutral or feebly alkaline after human milk, and slightly acid or alkaline after cow's milk. It is distinctly acid in 15 minutes, but free HCl is said by some observers to be only found in the fasting stomach. In the full stomach the acid is partly transformed into acid salts. Fatty acids are often present, lactic acid sometimes, and rennet ferment always. In suckling infants the contents are not infrequently streaked with altered blood, due to slight congestion from unusual digestive processes. Organisms are much more numerous in the bottle-fed than the breast-fed. They are insufficiently numerous to affect digestion, and vary little in number at different periods of digestion. The most frequent are *B. lactis*, staphylococci, *B. subtilis*, *B. butyricus* (if cow's milk is given), and *oïdium albicans* (if there is thrush).

In dyspepsia tough mucus is frequently present, together with lactic, acetic and butyric acids. The percentage of HCl may be abnormally high in subacute attacks. Atony is due to dyspepsia and persists for some days after symptoms have subsided. Food stays an abnormally long time in the stomach. Examination of the gastric contents after lavage affords information as to the nature of the contents, degree of digestion, and the mobility of the stomach. The conclusions drawn must not be dogmatically insisted upon as reliable.

**The Signs of Abdominal Disease** include not only those indicative of gastric disturbance, but also those of intestinal and peritoneal origin. Inspection may show umbilical peculiarities, general distension or retraction, dilatation of superficial veins, ecchymoses, peristalsis and the outlines of the stomach, small intestine and colon. The abdomen is normally prominent in the child up to about puberty, on account of the relatively narrow thorax, flat pelvis and large liver. The protuberance is exaggerated by intestinal fermentation and muscular weakness. Palpation reveals local tenderness, enlarged glands or tumours, and the position of normal organs. Sometimes it must be done in a warm bath or under anæsthesia. Percussion is always necessary and rectal examination frequently. Note alterations in the shape of the abdomen, unilateral swelling, general distension by gas (*tympanites*) and by fluid (*ascites*), and local distension.

The *Symptoms* include anorexia, flatulence, nausea, vomiting, hæmatemesis, alterations in frequency and character of the stools, and pain.



*Pain* may be of extra-abdominal origin. The pain of spinal caries may be referred to the epigastrium; that of pneumonia or pleurisy, especially diaphragmatic pleurisy, to the epigastrium or iliac fossa, and if on the right side it may be mistaken for appendicitis; and that of hip-disease referred to the iliac fossa. Rheumatic myalgia may produce paroxysmal attacks of pain in one or other hypochondrium. Intra-abdominal pain (*Gastralgia* or *Enteralgia*) is more commonly due to intestinal than to gastric causes. Gastric pain is due to hyperacidity, dyspepsia, inflammation and ulceration.

**The Appetite.**—Refusal of food is due to anorexia or dysphagia. Anorexia depends on lack of appetite, and this is present in febrile and toxæmic affections, in digestive and gastric disorders, and mere debility. Swallowing is difficult, painful or impossible in affections of the mouth, fauces and pharynx; in malformations of these regions; in dyspnœa due to nasal obstruction in infancy; from diphtheritic palsy and inco-ordination of the muscles of swallowing, possibly sometimes congenital; and in mental defects. Increased appetite (*Bulimia*) is produced by thirst, persistent over-feeding, acid dyspepsia, and occasionally by cerebral tumour.

**Pica**, dirt eating or depraved habit, is a curious perversion of taste which begins at 6-18 months of age, or even later. The child eats earth, gravel, coals, cinders, sand, paper, hair, wool, plaster, paint off toys and even fæces. All babies do so to a certain extent, through putting everything they get hold of into the mouth. The habit is due to a neurotic heredity, mental defect, lack of normal instinct, hallucination of taste, or alimentary disturbance and worms. It is increased by ennui, and may be limited to a single article. When indulged in to excess or in variety, it causes ill-health, wasting, sallowness, anæmia, colic and diarrhœa. If the habit is inveterate, it indicates a morbid perversion of taste, but must not by itself be regarded as proof of imbecility. It tends to spontaneous recovery in the third or fourth year of life, when the interests widen, though it may persist up to puberty or adolescence. Keep the child away from the desired article. Attend to the digestion and general health. Remove worms. Change the surroundings and keep the patient's mind occupied and interested.

**Vomiting.**—True vomiting is accompanied by pallor, nausea, facial distortion and violent effort. In functional or nervous vomiting the contents of the stomach, usually fluid, are brought up without effort. *Regurgitation* or *Over-flow Vomiting* in infancy is due to overdistension of the stomach, followed by contraction. The food is brought up in mouthfuls, without effort and without nausea, a few minutes after it has been swallowed and perhaps for sometime after. At first it only occurs immediately after feeding and the food brought back is unchanged. Later, it may be altered, sour and acrid, and contain curds, mucus and even bile. All the signs of infantile dyspepsia ensue, viz. pallor, restlessness, pain,



flatulence anorexia and malaise. The stomach is dilated and its motility impaired. Organic acids and bacteria are increased, and free HCl is absent. Regurgitation is also induced by eructations of gas, by pressure on the stomach, and by up-and-down jogging movements after food. Sometimes a whole feed is regurgitated at once, the result of it being swallowed too quickly. Regurgitation is associated with constipation at first, and later on with diarrhoea, often green stools, from fermentation. Uncontrollable vomiting in infants may be due to some undiscoverable quality in the mother's milk. *Habit Vomiting* is an exaggerated kind of regurgitation which may persist for years. Occasionally it is associated with chronic dyspepsia and a dilated stomach. In some respects it is analogous to rumination. It is painless, quiet and easy, without nausea, loss of appetite or gastric affection. Some babies seem able to make themselves sick on the least provocation, and are difficult to get on to more solid diet because of it. They may have to be fed many times in succession, although each feed is vomited, in order to overcome the habit. Such babies are neurotic, excitable children who have been rather badly managed. A trained nurse may be necessary for some days. The treatment for regurgitation in its mildest form consists in reducing the quantity of each feed and giving it more slowly. Starvation and lavage are only necessary if there is secondary gastric irritability.

**Rumination or Merycism** is a variety of regurgitation in which the food is returned into the mouth, after a meal, in definite and limited amounts, and is again chewed and swallowed. "Merycism" is derived from a Greek word, meaning "to chew the cud." The first case was described by Fabricius ab Aquapendente, in 1618. It has been said to be associated with rudimentary horns on the forehead. Out of 100 cases, collected by Sachs, of such horns only one was a ruminant. It occurs in all classes; is more or less hereditary (5 generations; Brockbank, 1907), and affects both sexes. It generally dates from early childhood or infancy, or does not begin until 10-20 years of age. It has been acquired by imitation, and has followed infectious disease and stomach troubles. It is an involuntary act, sometimes under the control of the will, beginning a few minutes after a meal and continuing for an hour or more. It may be influenced by diet, and is most apt to follow meat and eating too quickly. The process is quite pleasant unless the stomach is out of order.

*Vomiting* is a symptom of many conditions and is caused by direct action on the gastric mucosa, reflex stimulation of the vagus centre in the medulla or direct stimulation of it by the blood, alterations in blood pressure and by impulses from higher parts of the brain, e.g.—

- (1) Unsuitable ingesta. Overfeeding. Dietetic errors.
- (2) Inflammatory affections and functional disorders of stomach, intestines and peritoneum.



## (3) Obstructive disorders of the alimentary canal :—

- (a) In the lumen—foreign bodies, impacted fæces, worms.
- (b) Stenosis or atresia.
- (c) Intussusception, volvulus, hernia.
- (d) Pressure on the gut from without.

## (4) Toxæmia :—

- (a) Uræmia. Ptomaine poisoning. Febrile states. Anæsthetics.
- (b) Diphtheria. Acetonæmic states (q.v.).

## (5) Febrile Vomiting—chiefly toxic, partly due to arrested digestion.

## (6) Mechanical—due to paroxysmal cough.

## (7) Nervous :—

- (a) Functional—Habit vomiting, hysteria, anorexia nervosa, migraine (?), recurrent vomiting (?).
- (b) Organic—Tumours, meningitis, hydrocephalus, concussion.
- (c) Reflex—Pharyngeal irritation, teething, irritants in stomach and intestines, intestinal obstruction, passage of calculi, worms (?).

## (8) Excessive heat or cold.

## (9) Offensive sights or smells, and violent emotion.

## (10) Disturbances of equilibrium :—

- (a) Vertigo, swinging, mal de mer.
- (b) Travelling by rail, etc., especially with the back to the direction of progress.

The treatment of vomiting is primarily starvation. Other measures are lavage, counter-irritants, drugs and nutrient enemata. Recourse may be had to bismuth and alkalies, dilute HCl and nux vomica, cocaine gr.  $\frac{1}{100}$ – $\frac{1}{10}$  in dr. 1 of water hourly, creosote gr.  $\frac{1}{20}$ – $\frac{1}{10}$  with sugar and aq. anethi two hourly, chloretone gr.  $\frac{1}{2}$ , tinct. iodi m. 1–5 in glycerine and water hourly, vin. ipecac. m.  $\frac{1}{8}$ –1 or liq. arsenicalis m. 1 before food, resorcin grs.  $\frac{1}{4}$ –2 every two hours, strong black coffee, sips of very hot water or iced water. Apply a mustard leaf to the epigastrium.

*Lavage.*—Use a soft rubber No. 10–14 catheter with solid end, and an internal diameter 5 mm. and external diameter 8 mm., attached to a rubber tube about 2 feet long and a funnel. Pass it through the mouth, not the nose, into the stomach and irrigate with boric acid or sodium chloride dr. 1 to lukewarm water 1 pint, or benzoate of soda 1–2 per cent. strength. Introduce the fluid slowly. Empty the stomach and repeat the process until the fluid returns clear. The baby must be well wrapped up, including the arms, for lavage often causes the bowels to act. Take the child on the lap, and support it with the left hand and arm while



passing the tube with the right hand. For older children the sitting posture and a gag may be necessary, and two people, one holding the child with the legs between the knees, and the head steadied by the left hand on the forehead and pressed against the chest. Careful lavage is very rarely injurious. In rare instances it has caused cessation of breathing and convulsions.

**Gastric Digestion.**—Gastric juice in babies has a relatively high acidity and recently Sedgwick has found in the stomach contents a ferment, *lipase*, which splits up milk fats into free fatty acids producing this high acidity. Ibrahim (1909) has found lipase in the gastric contents and mucosa. The secretion of HCl is decreased or suppressed in slight disturbances of digestion and in ill-health. Normally in the breast-fed babe free acid appears about  $1\frac{1}{4}$  hours after a meal; in the bottle-fed, 2 hours. Though secreted immediately, it combines at first with the protein and alkaline salts. These substances are greater in amount in cow's milk and, therefore, the acid is longer appearing in the free state. The combined acid has no bactericidal power, but free acid has an inhibitory effect on microbial growth. Consequently micro-organisms will grow more readily in the bottle-fed than in the breast-fed. The intervals between meals should be sufficiently long to permit the secretion of free acid; and longer in the bottle-fed infants. The administration of diluted HCl after a meal is sound treatment for the prevention of microbial growth.

Hyperchlorhydria has not been sufficiently investigated to afford reliable proof of the actual existence of an excess of HCl. The total acidity is an insufficient test, for it may be due to lactic and other organic acids. The acidity of the gastric juice is decreased by any condition which increases the secretion of mucin. The rennet enzyme is always present and pepsin is rarely deficient. The action of rennet ferment on cow's milk differs from that on human milk. The latter does not clot at all unless a little HCl is added, and then it only forms a fine flocculent coagulum. Fresh undiluted cow's milk forms a coherent clot but, if it is sufficiently diluted and agitated, the clot is similar to that formed in human milk. Biedert states that the coagulation of cow's milk in the stomach is similar to that of human milk, if the ratio of fat to protein is the same. Dilution with egg albumin, gelatine or a cereal decoction has a similar effect on the clot. It is dependent on the degree of dilution and reduction of acidity rather than on the nature of the diluent. Some chemists have ascribed to rennet the power of dehydrating peptone and converting it into an albumin. The change is said to take place in the mucous membrane of the stomach and intestines, the various products of protein digestion being converted into a substance named "plastein," from which protein tissues are built up. According to Rotondi the rennet ferment acting on casein splits off a protein body, which is soluble in acetic acid and is not coagulable by heat.



The general conclusions from the study of gastric digestion in babies are that rennet ferment is always present; that pepsin is almost invariably present; and that HCl acid is often decreased in amount, sometimes is entirely suppressed, and occasionally is excessive.

**Dyspepsia.**—In dyspepsia or gastric indigestion the secretory function may be defective. This can be estimated by test meals, lavage and chemical tests. The gastric juice or the HCl is at fault. Dietetic treatment depends on the kind of disturbance. Egg-white, starch and sugar are inert, milk and gelatine little stimulating, and fat actually inhibits secretion. Stimulants of secretion include salt, meat extracts, condiments, spices, alcohol, and palatable food generally through its psychical effect. Bitters are of little use and are generally disliked. Equal parts of the tinctures of nux vomica, orange peel, gentian and aniseed are sometimes well taken, and useful as a stimulant for the appetite. One drop in water should be given to babies before breakfast and dinner. Strychnia gr.  $\frac{1}{100}$  can be given in syrup of liquorice. HCl or alkalies before meals are often useful. Bromides and belladonna reduce secretion. Burnt magnesia neutralizes hyperacidity.

If the defect is a motor one it is due to hypotonicity, defective motility the result of atony, or excessive motility set up by pyloric spasm. The spasm can occasionally be relaxed by local heat. Atony is treated by small meals of soft food, strychnia or nux vomica, mineral acids, alcohol, massage, douches and electricity.

Sensory disturbance causes hyperæsthesia and is treated by diet, heat locally, bismuth, hydrocyanic acid and bromides. Anorexia may depend on deficient sensibility.

*Dyspepsia in the Newborn* gives rise to slow digestion and the presence of curds in the stools, or the vomiting of curdled milk in  $1\frac{1}{2}$ -2 hours, when it should have passed through the pylorus. It is commonly due to debility, erroneous feeding, and dilatation of the stomach; and it sets up marasmus, if unrelieved. Lavage generally shows that the gastric juice is deficient in HCl. *Dyspepsia in Infants* is also due to dietetic causes or general weakness, frequently coincides with dilatation of the stomach, and ends in marasmus. In acute cases the infant should be starved for 12 hours, and then fed on whey, albumin water, barley water, weak tea, weak broth or malted milk. As the symptoms subside it is given peptonised milk, and finally milk and barley water. Teaspoonful doses of lime water and cinnamon water (p.a.) prevent vomiting.

*Acute Dyspepsia or Acute Gastric Indigestion* causes pain and discomfort, distension, eructations and vomiting. It is often preceded by fever and prostration; or by languor, discomfort and flatulence; and in infants by collapse. Vomiting occurs some hours after feeding and food, mucus and bile are brought up. The chief predisposing factors are general delicacy, rickets, liability to catarrh, chills and teething. The exciting causes are stale and indigestible food, foreign bodies, drugs, and exaggerated



irritability of the mucous membrane. Overfeeding in infants and too rich food are common causes. Some attacks follow exposure to cold. The nervous symptoms are restlessness, dulness or stupor, and convulsions which may be fatal in infancy. The pulse is weak, pupils sometimes contracted, and prostration severe. The tongue is furred and appetite lost. Fever rarely lasts more than a few hours, but may be very high. Attacks may be followed by gastric catarrh, colic and diarrhoea, or chronic dyspepsia. Infants waste rapidly.

General treatment consists in an emetic of warm water or lavage, heat or counter-irritants to the epigastrium, and an aperient of grey powder or calomel, with temporary starvation and regulation of the diet. The child is quite well in 1-3 days, but is liable to relapses from trivial causes. Return to ordinary diet must be gradual.

*Chronic Dyspepsia in Infants* is a common infantile ailment, especially in congenital syphilis, tuberculosis and marasmus. Improper diet is the great cause. The tongue is furred and, later on, becomes red and dry. Thirst, wasting and constipation are frequently present, together with regurgitation of food, acid eructations, vomiting, excess of mucus in the gastric secretion, abdominal distension and pain. The infant is restless, has a variable appetite, frequently develops thrush, and suffers from general malnutrition. The disease may terminate in exhaustion, anæmia, sudden collapse, thrombosis, convulsions and coma, just as in marasmus from other causes. Apart from prevention the treatment depends upon good hygiene and nursing, daily lavage, small suitable meals, and the administration of HCl. Possibly pepsin is beneficial, and sodium salicylate if there is much flatulence.

*Chronic Dyspepsia in Older Children* is often overlooked or ascribed to more serious disease. Vomiting is uncommon, constipation predominant and wasting considerable. It is especially apt to follow chills from insufficient or badly applied clothing, notably bare legs and arms. The secondary indirect or reflex symptoms may be so prominent as to mask the true cause. Some children come under observation for headache, especially in the morning, giddiness, disturbed sleep, pavor, teeth grinding and mental depression. Palpitations, cardiac irregularity, and anginal attacks raise the fear of heart mischief. In other cases an evening rise of temperature and dry hacking cough suggest early phthisis. Flatulent distension may be great, cause pain, and give rise to resistance on examination. If it is combined with fever, wasting, and alternate diarrhoea and constipation, it suggests tuberculous peritonitis, but the doughy feeling of the abdomen is absent. The association of headache, mental apathy, teeth grinding, constipation, vomiting and irregularity of the bowels is suspiciously like the onset of tuberculous meningitis. More especially is this the case, if there are fits. Night terrors are common. Sometimes there are syncopal attacks. Petit mal must be carefully excluded. Occasionally asthmatic attacks, with



cyanosis and rapid breathing, are entirely due to gastric catarrh. Often the symptoms are ascribed to worms, which may be present because of a chronic catarrhal condition of the intestines.

There are two types of chronic dyspepsia : (1) *Gastric* ; (2) *Intestinal*. Both may be present in the same child. It is often difficult to distinguish between them, and the symptoms as described above are partly those of gastric conditions and partly those of the intestinal affection. The latter disease will be considered more fully later. In the differential diagnosis considerable stress must be laid upon the presence of the furred tongue, discomfort after meals, and gastric dilatation in chronic gastric dyspepsia ; and the absence of these symptoms in that of intestinal origins. Gastric dyspepsia is very frequent in marasmic infants, but comparatively rare in older children, except as a purely temporary condition lasting a few days or a complication of more serious illness. On the other hand intestinal dyspepsia is seen at any age and is common in childhood.

Simple measures of treatment are sufficient for most cases. Light diet, fresh air, exercise and hygiene are the ordinary measures adopted. Grey powder and rhubarb are given every 2 or 3 days ; and a mixture of sod. bicarb., nux vomica and tr. rhei, or of gentian, soda and rhubarb, 3 times a day before food ; or a HCl mixture after meals. Bicarbonate of sodium in hot water or Vichy water, on rising and an hour before meals, washes out the stomach and dissolves mucus, and thus improves the digestion. No syrups should be prescribed because of the liability to fermentation.

*Achylia Gastrica* is comparatively rare before puberty. It gives rise to general malnutrition, abdominal pain and chronic diarrhœa, though the appetite remains good. The stools are liquid or pultaceous, foetid and clay coloured. They contain no starch, exceedingly few fat droplets, small white granules of fatty acid needles, little connective tissue, undigested meat fibres and trypsin. Examination of the gastric contents after a test meal shows absence of rennet and HCl, and that pepsin is either greatly deficient or absent. The affection is a disturbance in digestion of meat, but not of fat.

**Acute Gastritis** is catarrhal, ulcerative or membranous. The acute catarrhal variety is due to cold, unsuitable diet, alcohol and irritants. It may come on at any age, even in babies, and is sometimes set up by champagne. Occasionally small follicular ulcers are formed (*follicular gastritis*). The symptoms are those of acute dyspepsia, but are more severe and prolonged. The temperature is often high and the vomiting constant.

Furred tongue, anorexia, thirst, languor and debility, and epigastric pain and tenderness are present, just as in acute gastric dyspepsia. But the vomiting is more persistent, the thirst more intense, and the local signs are exaggerated. Sometimes there is profound collapse. The



abdominal pain and tenderness may be so severe as to suggest peritonitis. The fever and acute symptoms are worst at the onset and subside in a day or two. The usual duration of a properly treated attack is 3-7 days, but it may be prolonged by bad management, too early return to ordinary diet, and end in chronic gastritis. The *ulcerative* variety is secondary to a *phlegmonous gastritis*, with necrosis of the mucosa, set up by corrosive poisons or infectious diseases, such as smallpox, measles, scarlatina, etc., and pyæmia. The necrosed mucosa may be vomited. It is generally fatal, if due to corrosive poisons. The treatment of acute gastritis is by alkalies bismuth, and the methods adopted for acute dyspepsia. For corrosive poisoning the proper antidote is given and then oily fluids, milk and albumin water; morphia sub cutem; but no lavage.

*Membranous Gastritis* is characterised by the formation of a false membrane over all or part of the mucosa. The membrane is composed of exudation, fibrin, blood cells and microbes. A boy, 18 months old, admitted under my care for rickets and constipation, developed bacteriuria, albuminuria, nasal discharge, debility and vomiting. A small dirty yellow patch was present on the tonsils for two days before death. The œsophagus contained much membrane in the upper third. In the stomach almost surrounding the pyloric half, but not quite reaching the pylorus, that is in the pyloric vestibule, was an intensely engorged hæmorrhagic area, partly covered with adherent yellow membrane. Diphtheria bacilli were recovered from the œsophagus and the stomach.

A false membrane in the stomach has been recorded in about 12 cases of diphtheria, 4 of pneumonia, and 2 of pulmonary phthisis. Diphtheritic inflammation is probably always secondary to diphtheria of the throat or respiratory tract. It occurs almost exclusively in children but has been reported in young adults. It produces isolated patches in the fundus; streaks of membrane radiating from the fundus to pylorus; and, rarely, extensive ulceration or a universal false membrane. Deficiency of HCl may explain the preponderance of cases in the young, and the fact that separation of the membrane takes place least rapidly at the pyloric half. Probably all other cases of membranous gastritis are pneumococcal, though a few may be due to other organisms or to aphthæ.

Symptoms may be absent, or variable in severity up to distressing thirst, frequent and uncontrollable vomiting and bad epigastric pains, grafted on those of the primary disease. Casts or shreds of membrane may be vomited. Apart from this, the affection cannot be diagnosed, and usually the gastric symptoms are put down to the severity of the primary disease. The prognosis is bad. Grünbaum's (1902) case, probably diphtheritic, recovered. The treatment is that of acute gastritis; allaying vomiting and feeding by rectum.

**Hair Balls** are not uncommon in the stomach of animals which lick themselves, such as cows, lambs and cats. They are rare in human beings,



and usually, though not invariably, in girls or women with weak intellects. The habit of pulling out the hair may develop as early as the fifth month of life. Pernet has given it the name of "*trichotillomania*." The hair is swallowed and by the action of the stomach is felted into rounded masses of variable size, some being small enough to pass through the pylorus and per anum. They are called *ægropiles*. They may form a complete cast of the stomach. The symptoms are those of dyspepsia, of colic and diarrhœa, or may be entirely absent; the hard lump in the stomach being discovered quite accidentally. Sometimes there are vomiting and anorexia. Analogous habits are those of eating wool off blankets, chewing grass or paper, and "pica" or dirt eating. The diagnosis is easy, if the condition is suspected and the habit known; otherwise the tumour may be mistaken for something more serious.

**Gastric Ulcer.**—*Simple erosion*, due to hæmorrhagic extravasation, is not uncommon. The mucous membrane often exhibits hæmorrhagic patches, with blackish centre and irregular edges, and numerous spots of ecchymosis. Minute *follicular ulcers*, circular and multiple, are caused by necrosis of solitary glands. They are common in infants with follicular gastritis, frequent in children, and rare in adults. Hæmatemesis and melæna neonatorum may be due to erosions or follicular ulcers. *Diffuse ulceration* is produced by scalds, corrosive fluids, poisons and diphtheria. Severe scalds cause much destruction and are rapidly fatal, while slighter ones may induce cicatricial contraction.

*Tuberculous ulcers* are of two types. Some are minute, round, with thickened edges, and perhaps multiple; others are large and irregular. The small ones are probably due to a blood infection, and may be found in any fatal case of tuberculosis. The large ones are rare. The small ones are unimportant.

The *simple peptic ulcer* is acute or chronic. An acute ulcer is circular or oval, sharply defined, more or less perpendicular, sometimes funnel-shaped, with soft edges, no surrounding inflammatory thickening and a base in the muscular or serous coat. The chronic ulcer is large, irregular, with sloping and indurated edges, and a base often adherent to subjacent structures. Chronic ulcers are most frequent at the pyloric and acute ones at the cardiac end. They may be on the anterior or posterior wall; on the lesser or greater curvature. In number they vary from one to many, usually single, and in size from  $\frac{1}{8}$ -1 in. or more. Under the age of 10 years both sexes are equally liable. The simple ulcer is most common in the first two weeks, and rare after the first two years of life.

**Etiology.**—Some cases are congenital, due to a natal or ante-natal cause. Charcot found 2 ulcers in the stomach of a 6 months fœtus affected with smallpox. Asphyxia at birth may induce congestion of the mucosa and secondary necrosis or hæmorrhagic erosion. Subsequently, an ulcer may be due to an acute infection. Thus it has been found in infectious disease, such



as varicella, measles, typhoid, smallpox, diphtheria, noma, empyema, pneumonic affections, and after burns. Many of the recorded cases have occurred within the first few days or months of life.

*Symptoms* are often indefinite and irregular, and may be entirely absent, an unexpected ulcer being found after death. During the first month of life there may be hæmatemesis and melæna, or merely restlessness and signs of indigestion or gastric catarrh. After this, dyspepsia, pain and vomiting are more common. The pain is situated in the epigastrium, is paroxysmal, usually not severe, comes on after food and causes vomiting. It may be mistaken for colic. It is relieved by vomiting. Intense pain indicates bleeding or perforation. In older children the pain may be referred to the last dorsal or the first lumbar vertebra. It may be uninfluenced by food. Vomiting is most common if there is follicular gastritis. In older children it is preceded by nausea, occasionally by profuse salivation, and comes on when the pain is severe. Hæmatemesis is less frequent than vomiting. It may follow food, exercise or emotion. It varies in quantity, may occur without vomiting and give rise to melæna. The vomited blood is bright red, brown or even black. The tongue is generally clean, the appetite unimpaired, and the bowels confined. Sometimes there is chronic dyspepsia or gastric catarrh, with furred tongue, anorexia, flatulence, pyrosis and dilated stomach. Diarrhœa is present in about half the acute cases. Fever is not infrequent, because of the associated infection. The child becomes anæmic, emaciated and stunted. On examination of the abdomen a tender spot may be found in the epigastrium or at the tip of the ensiform cartilage, and in chronic cases some dilatation of the stomach. Perforation may be the first indication. The hæmatemesis must not be mistaken for that due to other causes, notably blood swallowed.

*Course and Prognosis.*—Except in infants fatal hæmorrhage is rare, but may occur even from follicular ulcers. Perforation and secondary peritonitis are rare in newborns. Fever and fits may be due to peritonitis. Cases have ended in adhesion to the diaphragm, perforation and empyema; in death from chronic malnutrition and asthenia, peritonitis, hæmorrhage, or some intercurrent disease. In Chvostek's case symptoms began at 4 months and the child died, small and emaciated, at 18 years. The stomach was enormously dilated and there was some pyloric stenosis, with a chronic ulcer near the pylorus and a recent ulcer near the cardiac end. Donner has reported a healed ulcer at 3 years of age. Probably 75 per cent. recover, though most of the cases on record are the fatal ones. Constitutional debility from any cause helps to produce an ulcer as well as prevent its cure. Adhesion to neighbouring organs is probably injurious by preventing contraction of the ulcer. Hyperacidity is not necessarily present, but is harmful.

Complications include dilatation of the stomach, pyloric stenosis, hæmorrhage, perforation, peritonitis, local abscess, and perforation of the



diaphragm. Both severe bleeding and perforation may be ushered in by great pain. Perforation is common in acute cases and not rare in chronic ones. It gives rise to sharp, sudden abdominal pain, collapse and vomiting, and may end fatally in two hours. In one case it caused death from convulsions and fever.

*The treatment* is similar to that of adults, and consists of careful diet and rectal feeding. The chief drugs are alkalies, calcined magnesia and bismuth, with the addition of small doses of morphia or opium for the relief of pain. The beneficial effects of horse serum, by mouth or rectum, on hæmorrhagic conditions in older patients render it worth a trial in these cases. Perforation is treated on ordinary surgical principles.

**Duodenal Ulcer.**—A considerable number of cases of duodenal ulcer have been found in melæna neonatorum, and a few in older children, but it is uncommon after the first two weeks and quite rare after the first year of life. Males are twice as often affected as females. The ulcer is usually single, situated in the first part of the duodenum just below the papilla, and of the simple type. Many are of septic origin or follow extensive burns. In a boy of 8 months hæmatemesis and perforation ensued on acute eczema becoming impetiginous (Borland, 1903). In a child, aged 10 months, the ulcer was a sequel of catarrhal colitis (Vanderpoel). Others are possibly due to a duodenitis, of septic or bacillus coli origin, or produced by hyperacidity of the chyme acting on a patch of hæmorrhagic infiltration or thrombosis.

Symptoms may be entirely absent. Restlessness, constant crying, meteorism, abdominal pain, the hands placed over the abdomen, and rapid emaciation may be noted in infants. There may be melæna, alone or associated with hæmatemesis. In older children the pain comes on about 2-4 hours after food, and is due to contact of the ulcer with the acid contents of the stomach. It extends round the right hypochondrium and through to the back. Sometimes a tender spot is found a little above and to the right of the navel. Constipation and anorexia may be marked. In other cases the appetite is good, pain is relieved by food, and the symptoms are those of dyspepsia. Perforation may be the first sign. Nephritis is not infrequent.

It may lead to duodenal stenosis, obstruction of the bile and pancreatic ducts, secondary disease of the gall bladder and pancreas, constriction of the portal vein, and local abscess. Operation is contra-indicated by *profuse* hæmorrhage, for such cases die in a few hours. The treatment is similar to that of gastric ulcer. Attacks may be followed by periods of latency.



## CHAPTER XXIII.

### THE PYLORUS.

#### *Pyloric Spasm—Congenital Hypertrophic Stenosis.*

Atresia of the pylorus is almost unknown. Simple congenital stenosis has been put forward as the explanation of gastric dilatation, dyspeptic symptoms and vomiting, which have started in infancy and persisted into adult life; the pyloric canal being found abnormally small at operation. Such a small canal might be blocked by a plug of inspissated mucus or swollen mucous membrane in gastric catarrh. Hall (1906) reported the case of a child, aged 7 months, who had persistent vomiting from birth and died after gastro-enterostomy. A plug of mucus due to chronic gastritis blocked the pylorus. The intestines were empty. Pyloric obstruction, due to enlarged glands, dermoids or new-growth; to polypus of the mucous membrane; and obstruction by impacted food or foreign bodies, are so rare as to be practically negligible in children.

**Pyloric Spasm** is characterised by severe and continued vomiting. Usually the food is brought up at once, but a considerable quantity may be kept down and then violently ejected. This type of vomiting may begin shortly after birth, or even some months later, and continue although the child is apparently properly fed. It is generally started by improper feeding. A typical case was that of a breast-fed girl who began vomiting three days after birth. Vomiting persisted, with constipation and wasting, for 11 weeks. She was then anæmic and emaciated, but no dilatation of the stomach, peristalsis or palpable pylorus could be demonstrated. She died the next day, although she had retained 26 oz. of whey. The pylorus showed no trace of stenosis or hypertrophy. Similar cases may start at any period during the first year of life, but in my experience are uncommon after the third month.

The etiology of the affection is doubtful. It is probably due to hyperæsthesia of the pyloric mucosa, started in the first instance by unsuitable food or local erosion, and the spasm is secondary to the hyperæsthesia. These cases are curable by a diet of whey or albumin water, and frequent small doses of cocaine. In one instance it was necessary to give the child cocaine and teaspoonful feeds of raw meat juice every  $\frac{1}{4}$  hour for 2 days, before the vomiting stopped. This was a boy, 7 months of age, who, while breast-fed, began vomiting in the seventh week of life and continued



until breast-feeding was entirely omitted in the fifteenth week. The severe attack might be regarded as a recrudescence of a milder one in earlier life.

The most important point in these cases is the complete absence of pyloric stenosis or hypertrophy on post mortem examination. In the only two fatal cases under my notice the pyloric canal was, if anything, larger than normal. Some of them may exhibit a dilated stomach and a moderate degree of peristalsis during life. The successful results of treatment support the hypothesis put forward as to their pathology.

**Congenital Hypertrophic Stenosis.**—This affection has been described under the names of the “*congenital gastric spasm*,” “*congenital pyloric spasm*,” “*congenital pyloric stenosis*,” and “*infantile hypertrophy of the pylorus*.” The name “congenital hypertrophic stenosis” is justifiable because the affection is congenital, hypertrophy or hyperplasia is the main feature, and it leads to stenosis. It is the hypertrophy that is congenital not the stenosis.

The first case in infancy was recorded by Williamson in 1841. In 1898 I collected 20 definite cases, including 2 of my own; and 4 years later, in a conjoint paper with C. T. Dent, we were able to collect over 50. Now there are probably 150 to 200 on record, and references in the literature to anatomical specimens from many others. Of these, 21 have come under my own observation, the diagnosis being verified at operation or after death. The affection is, therefore, by no means uncommon, and must be frequently overlooked.

**Symptoms.**—The characteristic features of a typical case are the age of the child, the vomiting, constipation, wasting, visible peristalsis, dilatation of the stomach and a palpable pylorus.

These babies are normal at birth; a few have been premature. Males preponderate to a remarkable extent. Only 5 out of 21 were females. They come under observation during the first 3 months of life. The symptoms may begin within a few hours or days of birth, or not appear until the third or fourth week; occasionally not till the second month; rarely or never at a later date. Usually they begin in the second week. They develop just as readily in the breast-fed as in those brought up in other ways. If, as often happens, partial bottle-feeding has been begun, vomiting is almost always ascribed to the diet. It is not uncommon for a child to start life as “a fine baby born,” to gain weight rapidly for 2 or 3 weeks, and then to begin vomiting. Lack of appetite is the first sign. The child does not take the breast readily or is very soon satisfied.

Vomiting is likely to be mistaken for regurgitation at first. Later on it becomes more characteristic and forcible, like that seen in older children. The contents of the stomach may be violently ejected to a distance of three feet. In typical cases two or more meals are kept down, and then the whole lot is brought up forcibly, gushing through nose and



mouth, or shot out as through a pump. It causes some pain, but the babe is more comfortable with the stomach empty. There is no nausea and food may be taken almost immediately afterwards. If the stomach is washed out twice a day, and it is already chronically dilated, there may be no vomiting. The absence of vomiting, in cases treated by lavage, is apt to be misleading. Vomiting is also modified by the degree and acuteness of the stenosis. In some instances there is little obstruction and no vomiting, if appropriate diet is given. In acute obstruction it may be as severe as in acute gastritis or intestinal obstruction, with hæmatemesis of coffee-ground material. The vomited matter consists of the stomach contents and is free from bile. It often contains mucus, from secondary gastric catarrh, and lactic acid or hydrochloric acid. If the stomach is washed out and a fresh meal given, the whole may be recovered at the end of two hours, though it ought to have passed onward into the intestines.

The tongue remains clean and the breath sweet, unless gastric catarrh is set up. The appetite is poor, except when the stomach is emptied. Then food may be taken ravenously. Pain is caused by over-distension, if food is given when the stomach is already full.

Constipation is marked and increases in severity. The stools are often devoid of faecal material and resemble brownish or dark green paint, or meconium. Occasionally they are tarry. They consist of epithelial debris, intestinal secretions, altered bile and perhaps blood. If the obstruction is not continuously complete, faecal matter is present, and the stools may be many and watery; the result of enteric catarrh set up by irritant stomach contents. It is quite common for large normal stools to be passed during the first week or two of life, and the constipation to then develop gradually.

The child wastes progressively, sometimes with extraordinary rapidity. The body surface is cold, temperature subnormal; the pulse small, frequent, and weak; and the general appearance one of whining lethargy with half-open eyes and sunken fontanelles.

Visible peristalsis is an important physical sign. It is best elicited by giving a fair sized meal, or "a comforter" to suck, by flicking the stomach wall with a finger, or by the application of a cold object. The stomach bulges forward, forming a swelling about the size of half a golf ball in the left hypochondrium, and the swelling flows over like a wave from left to right to the pylorus; and occasionally passes onward down the duodenum, showing a marked constriction and a momentary stoppage at the pylorus. The unyielding linea alba gives the stomach during peristalsis an hour-glass appearance. The peristalsis is most marked in cases of long duration. If the stomach is much dilated and the walls weakened, the wave does not cause so marked a bulging of the abdominal wall. A mild degree of peristalsis is often visible in the dilated stomach of emaciated children, and must not be mistaken for peristalsis of this type.



Dilatation of the stomach is present in advanced stages. It depends on the degree and duration of obstruction. In the early stages it is absent, for food is ejected as soon as the stomach is over-distended. Gradually the stomach becomes dilated and holds as much as 14 oz. in the third month of life. It bulges forward the epigastrium, and gives the lower half of the abdomen an empty or retracted appearance. The greater curvature is visible through the thin parietes and reaches the level of the navel.

The pylorus is palpable as a hard lump about the size and shape of a filbert, sometimes more rounded and feeling like a small marble. I am convinced that it does not alter definitely in hardness or become more palpable during peristalsis, though that portion of the stomach adjacent to it, known as the *pars pylorica ventriculi* or *pyloric vestibule*, may contract and give rise to the sensation of a tumour. In the adult the *pars pylorica* has been seen at operation to contract up into a hard tumour-like mass, even during deep chloroform narcosis, and such spasm may last for a few seconds only or continue for days, but there is no true hypertrophy. This is probably the explanation of some cases in which the pylorus is said to be palpable at one time and not at another. In infants operated on for pyloric hypertrophy, a wave of peristalsis may be seen at the time of operation to pass from the stomach to the pylorus without causing the least change in its consistency or appearance. The pylorus is felt about  $\frac{1}{2}$  in. to the right of, and  $\frac{3}{4}$  in. above the umbilicus, roughly midway between the umbilicus and the costal margin. Occasionally it is lower down and may be mistaken for an enlarged gland. Sometimes it is buried beneath a large liver, though even then it can generally be felt. It is more or less palpable in practically every case, though it is impossible to be absolutely positive in well-nourished infants. The examination must be conducted with warm hands, the tips of the fingers being gently pressed deeply in the pyloric region, and slowly moved up and down. In wasted babies it may be possible to grasp the lump between the fingers in front and the thumb behind.

*Varieties.*—Cases may be divided into three types. The majority are typical ones, such as above described. Rarely the obstruction is acute, probably in consequence of œdema of the mucous membrane of the pylorus, set up by rapid contraction of the hypertrophied muscle. In the third variety there is less hypertrophy and the muscle does not contract sufficiently to cause complete obstruction. Cases of this type may live for years, e.g., 4 years (Hezekiah Beardsley, 1788); 5 years (Sonnenberg); and 11 years (Hansy). Probably the hypertrophic stenosis of adults is due to persistence of the infantile condition.

*Morbid Anatomy.*—The normal pylorus is a constriction, the thickness of an old-fashioned wedding ring, formed by the circular muscular fibres. In hypertrophic stenosis it forms a hard, elongated, cylindrical tumour,  $\frac{3}{4}$ -1 in. long, and about  $1\frac{1}{2}$  in. in circumference; a solid tumour about the



size of the last joint of the female little finger. In advanced cases it is hard, white and bloodless. It terminates abruptly at the duodenal end, projecting into the duodenum much as the os uteri projects into the vagina, and looking like it when viewed from the duodenal aspect. On the gastric side it is more funnel-shaped, but ends abruptly. Microscopical examination shows that the enlargement is due to a great increase in the circular muscular fibres. I have examined many specimens and am convinced that all other changes are secondary. The mucous membrane is thrown into folds from 1-5 in number. Sometimes one fold projects as a ridge-like promontory along the whole length of the canal, in the same way as the verumontanum in the urethra. It may be congested in acute cases. It is to the folds of the mucous membrane that the complete obstruction is due ; and these folds are produced by the gradual or spasmodic contraction of the hypertrophied muscle. No stages of muscular hyperplasia are found intermediate between the normal pylorus, or the pylorus of infants who suffer apparently from pyloric spasm, and that of infants with true hypertrophy. Gastric catarrh, œdema of the pyloric mucosa and gastric dilatation are all absent at the onset of the initial symptoms.

*Pathology.*—It is probable that nature, in her extreme anxiety to provide an efficient pyloric sphincter, has produced too great a quantity of muscular tissue. In other words there is a true hyperplasia, a simple redundancy of foetal growth. The pylorus begins to be formed during the third month of foetal life, so there is plenty of time for hyperplasia to take place before birth. Such hyperplasia has been found by C. T. Dent in a 7 months foetus. Possibly it represents a reversion to an earlier type of pylorus, for the circular muscle is well marked in the pylorus of many mammals. It has been suggested that the hypertrophied pylorus is analogous to the “gastric mill” of crustacea, the “gizzard” of birds, and the “gastric mill” of edentata, closely resembling that of the great ant-eater and some armadilloes. The early appearance of symptoms and the degrees of hypertrophy present in cases dying within a few weeks of birth, seem quite incompatible with post-natal development. Operation within the first few days of life has shown the presence of as much hyperplasia as in the second or third month.

Thomson holds that “the essential lesion is not a muscular but a nervous one ; a functional disturbance of the stomach, and pylorus leading to ill-coordinated and therefore antagonistic action of their muscular development.” This hypothesis assumes the occurrence of ill-coordination ; it assumes that antagonistic muscular action and muscular hypertrophy are the result ; and it assumes that this nervous derangement can occur in foetal life. Thomson suggests that it is set up in foetal life by liquor amnii, which may be swallowed. It is difficult to understand why spasm should only occur and produce hypertrophy in the first three months of life and not at a later age. In the rare cases that come under observation



after the third month, the symptoms have dated from early infancy. Spasm will not explain the cases in which there is hypertrophy and little or no vomiting. Those adults, whose symptoms are put down to pyloric spasm, do not show evidence of pyloric hypertrophy at operation or after death.

There is no evidence that the spasm is set up by erroneous feeding or by hyperacidity. Observations by Miller and Willcox (1907) are of value in this connection. They found that in marasmus or atrophic dyspepsia, the acidity and ferment activity of the gastric contents was diminished, and that there was neither mucin nor retention of food in the stomach. In 5 cases of hypertrophic stenosis the acidity was variable and tended to be diminished, varying with the amount of gastritis. Ferment activity was markedly increased, mucin in excess, and food retained in the stomach. These results were absent in early stages, were modified by systematic lavage, and depended on secondary effects. In pyloric spasm or acid dyspepsia, there was neither mucin nor retention of food in the stomach; the ferment activity was normal or subnormal; and the acidity was increased. The test meals consisted of 2-3 oz. of diluted milk, withdrawn in 20 minutes; and the rennet activity of the filtered gastric juice was estimated by adding 0.01-0.5 c.c. to 5 c.c. milk at 40° C. These carefully conducted investigations are in favour of the view that spasm may be due to hyperacidity, and that in hypertrophic stenosis spasm, if present, is produced by some other cause, ? toxins of decomposing food. In some patients there is very little evidence of spasmodic obstruction; for vomiting is slight or absent, there is little peristalsis, and food passes readily through the pylorus. In such cases, on the hypothesis of spasm, we should be driven to assume that spasm had existed and produced a certain degree of hypertrophy, and that it had then ceased and left behind it a certain degree of mechanical obstruction. A more justifiable hypothesis is that there exists a considerable hyperplasia or hypertrophy, that this gradually contracts and produces obstruction, and that it may be exaggerated by attacks of pyloric spasm. Hence, in mild degrees of hypertrophy recovery may take place if spasm is absent or ceases under treatment. A hypertrophied pylorus does not necessarily contract sufficiently to cause serious obstruction.

There is no proof that spasm of the pylorus produces hypertrophy. In long continued anal spasm no hypertrophy results. Normally the pylorus is contracted and only opens, like a closed door, to permit the passage of chyme in response to the duodenal reflex, when the acid chyme which has passed through has been neutralized. Consequently very little extra exertion would be necessary to counteract the tendency to relaxation or to maintain contraction; certainly not sufficient to produce this great hypertrophy. Moreover, if spasm can produce hypertrophy, we ought to find post mortem evidence of all degrees of hypertrophy, up to the extreme amount present in these peculiar cases, in babies of all ages. Furthermore, few of these cases are amenable to medical treatment, even if they come under



observation at the earliest possible age. Anti-spasmodic drugs are unavailing. A large number of post mortem specimens accumulate under purely medical treatment.

*Diagnosis.*—Simple regurgitation, gastric catarrh, vomiting and constipation from unsuitable food, and habit vomiting must be excluded; so, too, rare forms of stenosis from other causes. The diagnosis often rests on a careful study of the history, symptoms and course of the illness, and the age of the patient. The presence of peristalsis and a palpable pylorus are the most reliable physical signs; but it is not necessary to wait until the pylorus can be felt with absolute certainty before coming to a definite conclusion. It is most important to distinguish pyloric spasm from hypertrophy. In spasm the vomiting is much less characteristic, peristalsis is slight or absent, the stomach rarely dilated, pronounced constipation unusual, diarrhœa may be present, and there is no tumour. Dilatation of the stomach is common in babies. If it is associated with vomiting, wasting and a certain degree of peristalsis, the diagnosis is very difficult. The presence of hyperacidity is in favour of spasm rather than stenosis. In case of doubt, the results of medical treatment are of assistance. On the whole the character of the vomiting, typical peristalsis, and complete constipation or meconium-like stools, are the most reliable signs in early stages. The presence of fæcal matter in the stools is in favour of spasm or of spasm *plus* hypertrophy. Stenosis cannot be complete and medical treatment may be given a further chance.

*Prognosis.*—A study of my cases affords valuable information about prognosis and treatment. One out of the 21 is omitted for it was only seen in consultation, subsequent to operation. All the 8 patients who were not operated on, died in the second to the fourth month of life. One of these died at the age of 14 weeks from enteritis. While under treatment there were periods during which he gained weight. A large almost normal stool was passed 8 days before death, and normal fæces were found in the intestines in considerable quantities after death. During the 10 days he was in hospital the pylorus was palpable, peristalsis was only observed once, the stomach was little dilated, and there was no characteristic vomiting. The appearance of the pylorus after death was quite typical. This case shows that hypertrophy alone does not necessarily produce obstruction. Of 5 private patients operated on at ages varying from 28-45 days, 4 recovered completely and have remained well. The fifth was treated on medical principles as the parents were opposed to operation, but eventually they decided on having the operation done as a forlorn hope. The child never rallied and died 17 hours later. Two other cases, seen in consultation and sent into hospital for operation, also recovered. Of the 5 remaining hospital cases 1 recovered, and died 3 months later from summer diarrhœa; another recovered from the operation and progressed favourably for a time, but succumbed to enteritis 54 days later. These



may justly be claimed as surgical successes and medical failures. A third died from the anæsthetic and shock ; a fourth died suddenly from syncope on the fourth day, while apparently progressing well, and no cause could be found post mortem ; and the fifth died from adhesive peritonitis 6 days after the operation, he had suffered from irregular pyrexia during the 9 days before he was operated on. In all but one, the too-late case, pyloroplasty was performed, and 8 out of 11 treated by pyloroplasty recovered. From the above results it is clear that the outlook is extremely bad if the treatment is purely medical, even though obstruction is incomplete. At a meeting of the Clinical Society of London (1906) Voelcker stated that out of 39 cases observed at the Hospital for Sick Children, Great Ormond Street, 34 died. Ashby, of Manchester, had made necropsies on 11 cases, only one of which had been operated on. Still ("Common Disorders of Childhood," 1909) claims 19 recoveries out of 42 cases. Of the 19 cured, 11 were treated medically. Of 20 fatal cases, 6 were treated by operation. Thus the operative mortality was 6 out of 14 ; three cases were lost sight of ; and 14 out of 25 died under medical treatment. John Thomson, who has done so much towards attracting attention to this disease ("Clinical Examination of Sick Children," 1908) states that he has seen 41 cases since 1894. Omitting one which was under treatment, his results were, 4 recoveries out of 17 treated medically, and 8 out of 23 which were operated on. In 3 instances no autopsy was obtained ; in 33 the diagnosis was verified at operation or after death. These results are very different from those obtained by Still, and support the view that it is difficult to differentiate between spasm and true hypertrophy.

The prognosis of operation depends chiefly upon the state of the child and the subsequent treatment. The greater the marasmus and dilatation of the stomach, the less is the chance of recovery. After successful operation the prognosis is that of the marasmus. It is quite possible that mild cases, and even some of considerable severity, may recover under medical treatment, if the obstruction is due to spasm grafted on hypertrophy rather than to hypertrophy alone. It is practically certain that many of the recorded cures are of this type or merely cases of pyloric spasm. Ibrahim, of Heidelberg, wrote me that two cases, which showed typical peristalsis, had recovered. One of these died 6 months later from pneumonia, and the hypertrophy of the pylorus was still present. In those few instances of recovery, in which the diagnosis of hypertrophy was certain, the prolonged and dangerous illness, apart from almost certain failure, far outweighs in risk the dangers of operative treatment. One might almost as well wait for an intussusception to be cured by spontaneous reduction or by sloughing, as postpone operation because instances of recovery have been recorded. After operation the child is practically well in 10-14 days. A child left with the condition unoperated on is for the remainder of life liable to further pyloric obstruction and dilatation of the stomach.



*Treatment.*—In a doubtful case put the child under careful observation and on a simple diet of breast-milk, ass's milk, whey, albumin water or Allenbury No. 1 Food. Give the food in small quantities at intervals of 2 hours. In addition give cocaine gr.  $\frac{1}{100}$ , or tr. opii. m.  $\frac{1}{8}$ - $\frac{1}{2}$ , every hour. If there is no improvement in 24 hours, wash out the stomach once or twice a day with a weak alkaline solution. This line of treatment may be continued for several days or even weeks, if the child is not losing ground. It is not advisable to persevere with it until the child is so wasted that the chance of recovery from operation is infinitesimal, although it has been asserted that the child goes on losing weight until 4 or 5 months of age, or is almost moribund, and then suddenly rounds the corner. Lavage is beneficial by washing away irritating gastric contents, the products of decomposition of food retained long in the stomach. If spasm is due to this cause, it may be relieved by lavage. One occasional complication, even in cases treated on these lines, is tetany; and even convulsions have occurred. These nervous phenomena are not unusual in chronic dilatation of the stomach. Lavage is further advantageous in helping to prevent over-distension of the stomach, provided that it is done systematically twice a day, and that small feeds are given in the intervening periods. It is certain that a greatly dilated stomach adds to the difficulties of treatment after operation; and it seems likely that such a stomach, full of liquid food, may drag on the pylorus and cause a definite amount of kinking, and thus increase the obstruction. This mode of treatment is almost certain to cure acid dyspepsia and simple spasm. It will relieve and assist in the cure of gastric catarrh. But it is difficult to believe that it can in the least degree affect the hypertrophy of the muscle fibres or be of the least value, except in those cases in which the obstruction is due to secondary effects, grafted on a primary hyperplasia of moderate severity. The choice of operation lies between pyloroplasty, Loreta's stretching operation, and posterior gastro-enterostomy. The stretching operation is somewhat analogous to forcible rupture of urethral stricture. For complete success it is necessary to rupture some or all of the circular muscle fibres, and the peritoneal coat is liable to be ruptured as well. This has been followed by fatal peritonitis. If the pylorus is merely dilated, it is liable to contract, and a second operation may be and has been necessary. Posterior gastro-enterostomy is satisfactory as regards the maintenance of nutrition, but does not affect the condition of the pylorus. Morse has reported a case, treated by this method, which died from another cause  $6\frac{1}{2}$  months later, aged 8 months. The pylorus was found in exactly the same condition as at the time of operation; a conclusive proof that spasm had nothing to do with the hypertrophy or the stenosis. Pyloroplasty is the most scientific method of treatment, for the pylorus can no longer contract after operation and there is no fear of recurrence. Its difficulties are overrated, for it can be done by a reasonably good surgeon in 20 minutes. It is important to have



a skilled anæsthetist. Immediately after the operation the child should be given a rectal feed of 2 oz. of peptonised milk and water, p.a., and 10-20 drops of brandy. Rectal feeds should be given every 4 hours for 2 days, every 6 hours for 2 days, and twice a day for 2 days. A teaspoonful of hot water must be given by mouth every  $\frac{1}{4}$  hour, while awake, for a period of 12 hours, and after that a similar quantity of whey. Subsequently the whey is increased to 2 teaspoonfuls every  $\frac{1}{4}$  hour, a tablespoonful every  $\frac{1}{2}$  hour, 1 oz. hourly, and finally 2 oz. every 2 hours. After that it is strengthened by the addition of cream; and gradually replaced by peptonised milk and water, or milk and water with cream, until the child is on an ordinary diet. Vomiting usually persists for 2-6 days, and some altered blood is brought up a few hours after the operation. There may be a little fever for 2 or 3 days. As a rule the vomiting subsides in a couple of days; food passes through the pylorus in 24-48 hours, and is found in the rectal wash on the third day; and the child is discharged cured at the end of a fortnight. Great care must be taken not to overfeed these children in the early stages of convalescence, on account of the liability to enteritis. This is especially apt to occur in marasmic infants, and is probably dependent upon nutritional changes in the intestinal mucosa, consequent on starvation. Œdema is another complication occasionally seen. It is due to overfeeding or to renal inadequacy.

Though recognising the possibility of cure in mild cases without operation, my experience leads me to assert that operation is the best possible treatment to advise and that, if done early, its risk is comparatively small. It is essential that the after-treatment is left in the hands of the physician, and not entrusted to the surgeon, for on this the life of the child is most likely to depend. There is grave danger of ascribing to pyloric hyperplasia the symptoms due to pyloric spasm only, and thus acquiring an unduly favourable idea of the prognosis in the more serious disease. An almost equivalent danger is that of diagnosing hypertrophy and operating, when the affection is purely spasmodic.



## CHAPTER XXIV.

### INTESTINAL DIGESTION.

*The Stools—Tympanites—Colic—Reflex Colic and Diarrhœa—Intestinal Dyspepsia—Carbohydrate Fever.*

**The Stools.**—Meconium is passed during birth, or a few hours after, and for a period of 1-4 days. If passed before birth it indicates sometimes, but not always, ante partum asphyxia. The total amount is 2-3 oz. It is odourless, homogeneous, viscid and tenacious, sterile, weakly acid, and semi-solid. In colour it is greenish, greenish-black, dark brown or brownish-black. It is composed of various intestinal materials, namely epithelium, mucus, bile and cholesterin, and the substances swallowed with the liquor amnii, such as squamous cells, fat globules and lanugo. The meconium corpuscles are oval or round, and greenish yellow; probably bile-stained altered epithelium cells. Bile begins to be secreted in the third month of foetal life. Meconium is found in the small intestine and appendix about the middle of the fourth month of foetal life, and in the large intestine in the beginning of the fifth month. After the seventh month it distends the whole large intestine. It has been found in the appendix although the cæcum was practically empty.

During the first 2 or 3 days after birth from 2-4 stools of meconium are passed. By the fourth or fifth day the golden yellow colour of ordinary milk fæces is acquired. Sometimes the meconial character persists for a week or more.

The normal amount of fæces passed daily by a breast-fed infant is 1-3 oz., or about 2 per cent. of the weight of milk ingested. The dried residue has been estimated at 15-20 per cent.; consisting of organic substances 90, and inorganic 10 per cent. One-third of the inorganic substances consists of calcium salts. The reaction is acid or neutral. In colour, consistency and general appearance, the fæces resemble the yolk of an egg, and they vary in colour according to the percentage of fat, as much as do the yolks of eggs, without being abnormal. The colour is due to bilirubin, and becomes greenish on exposure to air by conversion into biliverdin. The stools are mainly composed of fat; perhaps 25-35 per cent. of the dried solids is in the form of neutral fats, fatty acids and soaps. The acid reaction and slightly sour smell are due to these acids, chiefly lactic acid. The proteins of breast-milk are almost, but not entirely, absorbed. Mucus



and intestinal epithelium are present in considerable quantity, and the other constituents are bile pigments, cholesterin, lime salts and other inorganic salts, and rarely indol. Hydrobilirubin is not often found, because of the rarity of putrefactive intestinal changes. The gaseous discharges consist of hydrogen and carbonic acid gas. Sulphuretted hydrogen and marsh gas appear later in life. Micro-organisms are soon present in large numbers. The streptococcus acidilactici is the chief organism in the small intestine, and both it and *B. coli* are found in the meconium 10-12 hours after birth. This organism sours milk and restrains the growth of pathogenic bacteria. In the large intestine the *B. bifidus* is the most important organism. It is anaërobic and found in the stools after the meconial stage. The absence of a putrefactive odour is partly due to the rapid passage of the food through the alimentary tract; to the fact that milk does not usually undergo putrefaction, and a milk diet lessens intestinal putrefactive processes; and to the fact that fermentation has a similar effect. Putrefaction depends chiefly on the intestinal flora.

In infants fed on cow's milk the stools are larger in amount, equal to about one-fourth of the milk ingested, and contain casein. They are whitish or pale yellow in colour, and turn greyish yellow or white on exposure. The odour is cheesy or offensive, and the reaction alkaline. The addition of carbohydrates to the diet renders the stools more yellow in colour, acid in reaction, and free from the smell of decomposition. The acid reaction, due to the fermentative changes of carbohydrates, prevents putrefaction. The common organisms present are the *Streptococcus acidilactici*, *B. coli*, *B. lactis aërogenes* and *B. bifidus*, all of which cause fermentation but have no proteolytic or peptonising action.

Curds of two types are found in infantile stools. *Fat Curds* are small, soft, whitish lumps, sometimes pin-head in size, yellowish, and looking like yellow sandy particles or biliary concretions. They are composed of fat in the form of fatty acids and calcium soaps. They contain little nitrogen. *Casein Curds* are in the form of smooth white flakes, or large and tough lumps, and sink in water. They contain much nitrogen, little soap, and consist chiefly of casein. If there is no excess of casein in the milk given to infants, the stools may be the same as in the breast-fed. Gradually as the diet becomes more mixed, the stools acquire the adult characters, and become brownish and usually formed by the end of the second year. The healthy child passes 2-4 stools daily during the first fortnight and after that 2 a day, or a number varying from 1-4 or more. Occasionally the breast-fed infant passes a large number of normal stools, even 12 or 13 daily, and yet gains weight rapidly.

*Abnormalities of the Stools.*—They may be frothy; too liquid or too solid; acid or alkaline; variable in colour and odour, in size and in composition; and may contain parasites.

As a general rule, the paler the yellow colour, the less is the amount



of fat present. *Large colourless stools* may be due to failure of pancreatic digestion. They are acid and contain an excess of fat, chiefly fatty acid. Stercobilin is usually present and abundant though the stools are colourless. This condition is associated with a distended abdomen, anæmia, great and often rapid emaciation, and frequently a sore tongue, aphthæ and ulcers. Possibly there is pancreatitis of a mild type. Milk diet is useless. The child must be fed upon pounded beef, eggs, chicken, liver soup, jellies and barley water ; vegetables are added later. In some respects it is analogous to Sprue, for which santonin and ipecac. sine emetin are useful. In this affection also milk diet is useless, though milk and strawberries are beneficial. *White stools* have been ascribed to excess of milk curd ; to absence of bile, giving rise to offensive smell ; or excess of fat, causing an odour of rancid butter. Some infants overfed with cow's milk, but under-nourished, pass stools which are dry, neutral or faintly alkaline, very pale or even almost white, like those of acholia. These stools are not free from bile, but are the result of perverted liver function or change in the bile pigment. Bilirubin is changed into colourless urobilinogen instead of urobilin. With an acid solution of dimethylamido-azobenzaldehyde urobilinogen produces an intense red colour. Probably it is derived from hydrobilirubin in an alkaline medium, and the hydrobilirubin is due to putrefactive processes. Fæces containing the latter pigment turn red with a saturated solution of corrosive sublimate. If bilirubin is present it is converted into biliverdin, and the colour is green. Some of these stools are more the colour of clay or putty ; and perhaps due to a similar cause, acholia, an excess of starch, or gastro-intestinal catarrh. Starch can be recognised under the microscope or by its reaction with iodine.

Intestinal fermentation, if excessive, as it may be from excess of starch or carbohydrate food, gives rise to frothy stools with a sour smell. Intestinal putrefaction is due to decomposition of protein, and the stools are putrid and offensive. In all pathological states, especially catarrhal conditions of the intestine, the secretion of mucus is excessive. It is extraordinarily liable to decomposition, and produces an alkaline reaction in the stools and a foul smell. Clinically these effects are of great importance. Excessive or abnormal fermentation may be set up by unsuitable food, too large or too frequent meals, and give rise to fermentative dyspepsia with tympanites and the evacuation of numerous frothy acid stools. In moderation this process can be used as a curative agent, for the prevention of intestinal putrefaction and decomposition of protein. The lactic and other organic acids destroy or inhibit the growth of intestinal proteolytic and peptonising organisms.

*Green stools* may be green before or shortly after being passed, in consequence of the conversion of bilirubin into biliverdin by an alkali in the canal or the oxygen in the atmosphere. The colour has also been ascribed to the administration of alkalies by the mouth, and to intestinal



flora. More probably the conversion is due to an oxidizing ferment in the mucus or leucocytes. Wernstedt claims that he has identified such a ferment. The green colour is always associated with the secretion of mucus; the ferment action is secondary. *Slimy* or *mucoid stools* are those in which there is excess of mucus. If it is mainly from the colon, the stools may consist of little more than mucus. The stools may vary in colour from slatey blue or grey to black, due to the action of iron, bismuth, tannic acid or blood. They are often brown or greyish and offensive from the ingestion of raw meat juice; and may be bright red or black and tarry, due to blood from various parts of the alimentary tract. In rare instances membrane is present, for instance in membranous colitis. Worms and their eggs, parasites, pus, undigested cellulose, and various foreign bodies may be found and give valuable information as to the cause and nature of the illness.

*Examination of the Stools.*—Food passes from mouth to anus in 20 hours for lean meat (Strauss); 24 hours for mixed diet (Kozieskowski); 36-48 hours for milk diet (Maurel). Delay may be due to atony or a dilated sigmoid; and may occur in the stomach or small intestine, leading to auto-intoxication and attacks of bilious vomiting though the bowels act daily. Give one or two Belloc's pastilles of vegetable charcoal at breakfast; delayed appearance of the charcoal in the stools, if frequent, indicates that the diarrhœa is due to causes low down. In examining for occult blood exclude all hæmoglobin from the diet and make an ether extract of the fæces. Mucus is recognised by mixing the stool with water and examining it against a black surface. Undigested connective tissue, meat fibres, starch grains, ova of worms, etc., are found by microscopic examination.

**Intestinal Dyspepsia.**—The chief symptoms of intestinal dyspepsia are tympanites and colic. These may be associated with constipation or diarrhœa, lientery, and general malnutrition. Not infrequently there are nervous symptoms, either toxæmic or reflex in causation, such as irritability and fretfulness, violent and prolonged outbreaks of temper, teeth grinding, pavor and somnambulism.

**Tympanites.**—Distension of the abdomen, due to abnormal production of intestinal gases, is a chronic condition of gradual onset, or so rapid in its development as to deserve the name of "acute abdominal distension."

The chief causes are more or less associated in action, namely, excessive production of gas and intestinal muscular atony with imperfect peristalsis. Over-production of gas is due to bad feeding, carious teeth, imperfect mastication, bolting food, insufficient exercise, constipation, and fermentative processes. Intestinal atony is common in rickets, and in malnutrition from any cause, and is assisted by weakness of the abdominal walls. Imperfect peristalsis depends upon an insufficient supply of fluid and waste matter in the food, failure of pancreatic digestion, intestinal catarrh, abuse



of purgatives ; paralytic conditions from peritonitis, mechanical obstruction and nerve palsies, such as diphtheritic paralysis and affections of the spinal cord.

Acute distension is not very uncommon under 3 years of age. It is a serious sign in grave illness and often appears part of the act of dying. It is most often seen in diseases of the lungs and pleura, and greatly interferes with the breathing and the action of the heart, by upward pressure on the diaphragm. In older children distension is frequently chronic and the result of dyspepsia and bad feeding, often associated with constipation. The normally prominent abdomen of childhood must not be ascribed to this condition. Intestinal distension is general, except when arising from localised mechanical obstruction.

In chronic cases anti-fermentative drugs are given, but the treatment is mainly that of the local or constitutional state. Immediate measures are necessary in acute distension. The finger must be passed through the anus and the sphincter dilated if there is any spasm. An enema of soap and water, turpentine or asafoetida, is then given. The back and shoulders must be supported, strychnia injected, and brandy given by the mouth.

**Colic or Enteralgia** is paroxysmal intestinal pain from spasm of the intestinal muscles. In babies it is caused by flatulence from excess of protein, sugar or starches ; the pain being due to distension and spasm. In older children it chiefly follows the ingestion of hard or unsuitable food, or poisons such as lead. Vague pains are sometimes associated with worms, and may be referred to the navel or iliac fossa. Pain in the iliac region after exertion, such as hurrying to school after breakfast, may be due to chronic appendicitis. Colic is also set up by intestinal and peritoneal inflammation, appendicitis, intussusception, intestinal obstruction and scybala ; or it may arise simply from cold feet, chills, and drinking iced water. Infantile colic is characterised by moaning, screwing up of the face and paroxysmal cry ; and in bad attacks, by pallor, sweating, cold extremities and collapse. The knees are drawn up, the arms bent and the hands clenched. The abdomen is tense and hard, and the scrotum retracted. There is no local tenderness or fever. The attack is of short duration and relieved by the expulsion of wind. The cry differs from that of hunger in its loud and paroxysmal character, and it is associated with other signs of pain. The pain is lessened by food temporarily, but soon returns in probably a worse form. Simple colic must be distinguished from colic due to other causes, notably renal colic, intussusception and tuberculous adhesions.

*Treatment.*—The injection of warm water 2-6 oz., or glycerine dr.  $\frac{1}{2}$  in 1-2 oz. of cold water, generally leads to evacuation of wind and affords relief. During the attack apply warmth to the abdomen and feet ; give mild laxatives and carminatives, such as sp. chlorof., tr. card. co., tr. zingib., sp. vini. rect., dill water and peppermint in hot water ; small



doses of belladonna, hyoscyamus and codeia are useful for recurrent colic, or minim doses of Fowler's solution just before meals. Between the attacks attend to the digestion, avoid excess of starch, greens, fruit and vegetables containing cellulose. See that the clothing is sufficient and properly distributed.

**Reflex Colic and Diarrhœa.**—These names are more appropriate than “lienteric diarrhœa,” for they indicate the causation of the trouble and the appropriate lines of treatment. “*Lientery*” is derived from Greek words meaning “a smooth intestine.” It is defined in the New Sydenham Society's Lexicon of Medicine as “A species of diarrhœa, or looseness, in which the food passes rapidly through the bowel undigested, and nearly in the same condition as it was when taken . . . so called because the food seems to have slipped over a smooth-lined intestine.”

It occurs at any age, but more especially in the sixth year of life. This may be because dentition leads to bolting of food and imperfect digestion. It is quite common in infants, each feed being followed by an immediate action of the bowels. The presence of undigested food in the stools does not mean that the food has passed through the alimentary tract in the short time which elapses between ingestion and defæcation, but that it passes out of the stomach and through the intestines too rapidly to be properly digested, and consequently appears in an undigested form in the stools. Although gastric digestion is not essential in infancy, it is necessary that the food should stay sufficiently long in contact with the digestive juices, and not be hurried through the gut by unduly rapid peristalsis. Reflex peristalsis is set up as soon as food enters the stomach, is followed by griping pains or colic, and causes almost immediate evacuation of the lower bowel. The stools consist of undigested food and mucus.

These children are generally of the nervous type, excitable, neurotic, highly strung and variable in temper. Attacks are induced by food unsuitable in quantity and quality, given at too high or too low a temperature, or too rapidly. The habit of bolting the morning meal in order to hurry out to school or play, combined with sudden exposure to change of temperature on leaving the house and hurried bodily movements, is a common cause in children. In its simplest form it is induced by taking cold water or ice into the stomach, when heated. Colicky pains are at once induced and the bowels act profusely, with abdominal discomfort and even severe pain. Such attacks are easily relieved by warmth and temporary abstinence from food and drink. Reflex colic is more difficult of diagnosis when it is not severe enough to cause action of the bowels. It has to be differentiated from the colicky attacks of appendix origin, renal calculus, tuberculous adenitis or adhesions, gastric or duodenal ulcer, and such like causes. The prognosis is excellent. Untreated cases may continue for many months.



*Treatment* is simple. Food should be easily digestible, neither hot nor cold, and taken slowly. A certain amount of rest after meals must be insisted on. The child must be warmly clad, especially as regards the extremities; cold and wet feet being injurious. The most efficacious drug is Fowler's solution, m.  $\frac{1}{3}$ -1 in water dr. 1, before meals. Sometimes carminative digestive mixtures are required, if there is gastric catarrh. If these measures fail, recourse must be had to bismuth, bromide, codeia or small doses of Dover's powder. Bad cases are generally cured by change of climate and surroundings.

**Acute Intestinal Indigestion**, when sudden in onset, gives rise to fever for 12-24 hours. In children the temperature rises to 100-101° F., and in infants 102-105° F. Colicky pains are severe and cause screaming, restlessness, and drawing up the limbs. Tympanites, diarrhoea, prostration and nervous disturbance are commonly present. In older children the pain is situated about the umbilicus and followed in a few hours by diarrhoea. It is often diagnosed with difficulty from appendicitis. Recovery rapidly follows the administration of castor oil, a mercurial purge, or an enema.

**Intestinal Dyspepsia** has been described by Eustace Smith under the name of "*Mucous disease*," and by other observers as "*Lithæmia*," "*Neurasthenia*," and "*chronic dyspepsia in children*." These differences in nomenclature have given rise to much confusion, and it is doubtful whether the name of "intestinal dyspepsia" is sufficiently precise to include all the cases of this type. The characteristic feature is malnutrition or deficient growth, associated with alimentary symptoms and others of a more or less neurotic type. There is certainly some error of absorption or metabolism, not dependent upon stomachic conditions.

*Etiology*.—The predisposing factors are a family history of neuroses, gout, tuberculosis or digestive weakness; lowered general health from measles or whooping cough; overcrowding, bad hygiene, defective teeth, insufficient exercise, etc. Boys are more liable than girls in the proportion of about 2-1. Race, social position and residence have little effect. The age varies from 3-12, most commonly 5-8, the period of commencing second dentition. The teeth are often carious and septic matter is swallowed. Partly on this account and partly because of the age, food is bolted. The exciting causes are school life and an unsuitable diet containing an excess of carbohydrates.

*Symptoms*.—The onset may be acute with headache, vomiting and loose stools, but more often is indefinite. The child is languid, especially in the morning, is disinclined to get up, and "lays about" or "has no life in it." At night there is restlessness in the early part, teeth grinding, talking in the sleep, pavor, sometimes somnambulism and night sweats. This is followed by a deep lethargic slumber. The nocturnal restlessness is most common in school children and associated with constipation. In



contrast to the lethargy, these children are said to be very nervous, excitable, crying on the least provocation, starting at the slightest noise, afraid of being alone even in the daytime, and to blush or grow pale on the least emotional disturbance. These attacks of pallor are common and most characteristic. The child "comes over white," with black rings round the eyes, and the feeling of faintness lasts for several minutes but actual syncope is rare. The hands and feet are often cold, and the attacks may be associated with abdominal pain or worms. They are ascribed to enterospasm or intestinal distension, and they have to be distinguished from fainting attacks and petit mal.

More or less headache is generally present, usually frontal. It may occur at any time, especially in the morning or after school, and can sometimes be traced to visual defects. A slight or hacking cough, perhaps barking, worst in the morning, is often present and probably due to pharyngeal congestion. Pains are felt in the epigastrium or in either hypochondrium, and "stitch" in the side is frequent on exertion.

The appetite is lost, poor or ravenous. Vomiting rarely occurs except from exacerbations, which are usually described as "bilious attacks" or "attacks of gastric fever." They are ushered in by headache, vomiting and diarrhœa, anorexia, epigastric pains, high fever and even delirium. They vary in severity and duration, perhaps consisting only of vomiting and headache. Often they last for a few days, and are followed by a period of good health and constipation. Gradually the appetite becomes ravenous, more signs of the affection develop, and finally another acute attack. This description must be compared with that of recurrent vomiting (p. 158). Mild attacks are more prolonged than severe ones. The bowels are usually normal in appearance; occasionally grey and often very offensive. Sometimes the stools are large, offensive, and consist of scybala. Occasionally there is lenteric diarrhœa. Mucus is commonly present, if there is constipation or diarrhœa. Fever occurs in attacks in which the temperature ranges from 100-105° F., and together with the symptoms suggests typhoid fever. Gradually the child wastes, though this is not very noticeable to a stranger for the face suffers little.

On physical examination, the child is pale or sallow, may have a slight icteric tinge or actual jaundice, with dark rings round the eyes, and seems intensely languid or tired. On excitement he readily flushes up and looks quite healthy. Generally nutrition is poor, but the wasting rarely amounts to emaciation; the muscles are soft and flabby. The tongue has a whitish yellow fur far back on the dorsum and anteriorly is covered with mucoid secretion. It may be generally white and sodden, with prominent fungiform papillæ. The teeth are usually carious. Large tonsils and adenoids are present in about half the cases, but it is doubtful whether they have anything to do with the disease. Chronic pharyngitis is almost constant. The chest is often flattened, shoulders round, abdomen pot-bellied, and the



liver somewhat enlarged. The urine contains an excess of urates. Sometimes there is cyclic albuminuria. All varieties of worms have been noted, but thread worms are the most common. There is no fever except during the acute attacks.

*Carbohydrate Fever*, food fever, acute febrile or recurring gastric catarrh, is almost certainly one of this group of cases and does not require differentiation as a special entity. The name has been given to attacks of fever in children with more or less digestive disturbance, liability to recurrence, and no apparent origin. Some of them have been traced to excess of carbohydrate diet, and have been cured by strict limitation of this food stuff. The symptoms are an impaired appetite, more or less headache, general malaise, occasionally collapse, and fever for one to a few days, sometimes reaching a maximum of 104° F. Possibly the cause lies in the products of fermentation, a kind of toxæmia; or some catarrh is set up and the metabolism of the liver is impaired. The catarrhal hypothesis is unlikely to be true for a mercurial purge often cuts short the attack.

*Pathology.*—Intestinal dyspepsia has been ascribed by Eustace Smith to “A mucous flux which interferes mechanically with ingestion and absorption of food.” On the other hand, in many cases, no excess of mucus has been noted in the stools; more probably it is a result rather than a cause. The character of the stools in some patients, the lithuria, altered appetite and wasting show that there is a defect of absorption and assimilation. It has been ascribed to lithæmia or the uric acid diathesis, and undoubtedly many of the symptoms of lithæmia are due to intestinal dyspepsia. It has also been put down to auto-infection; and to over-stimulation of the glyco-genic function of the liver, because of the hepatic enlargement.

The most probable explanation is that these cases are due to errors in diet, chiefly an excess of carbohydrates combined with a deficiency of proteins, and that a catarrhal condition of the alimentary tract is set up, varying in severity. Consequently the secretions are altered in character, the food is not properly digested, by-products are formed, and there is imperfect absorption and assimilation of normal food stuffs, together with auto-intoxication from the absorption of the by-products. The lowered vitality and the catarrhal condition of the intestines permit the development of worms, and account for symptoms such as albuminuria and those due to impaired nutrition of the nervous system.

*Diagnosis.*—This affection has to be differentiated from tuberculous disease, especially the abdominal forms. It may occur as a complication of tuberculosis. The presence of adenoids and enlarged tonsils is too common in children of this age to have any bearing on the diagnosis. By interfering with breathing they may accentuate the general malnutrition and the symptoms, and account for some of the occasional complications, such as enuresis. Acute attacks are apt to be mistaken for appendicitis or other abdominal mischief. Headache and vomiting may be



mistaken for migraine; and the general symptoms ascribed to neurasthenia rather than the neurasthenic symptoms to the disease.

*Prognosis.*—The fever may last as long as 10 days and at times subsides suddenly. Recovery is rapid, except in recurrent cases. If untreated, it may continue for years in attacks of varying severity. It does not prove fatal in itself, but it lowers the general health and decreases the resisting power to other diseases. Relapses are liable to occur. Regularity of the bowels and the absence of mucus in the stools are favourable features.

*Treatment.*—For acute cases give a mercurial purge and simple diet, and rest in bed until 2 days after the fever has subsided. For chronic cases change of air and surroundings is more important than diet or drugs. Forbid all mental work, worry, excitement and entertainments. Keep the abdomen and extremities warm and dry. Attend to the teeth and fauces. Adenoids and large tonsils must be removed.

The diet consists of regular meals. Reduce or omit fermenting foods, sweets, jam and carbohydrates generally. Forbid all new bread, new potatoes, fried fish, pickles and stringy vegetables. Allow oatmeal porridge and stale brown bread tentatively. At first limit the diet to eggs, fish, bacon, ham, tongue, meat, potted meats, butter and dripping, dry toast and rusks. Later on, add greens and ripe fruit.

Cod-liver oil and hypophosphites are injurious unless the child is almost convalescent, and even then they may upset the digestion. Give grey powder, with or without rhubarb and soda, at bedtime; in small doses if there is diarrhoea, and larger doses if the bowels are constipated. Apeuta water or sodium sulphate on waking is necessary, if there is much constipation. A mixture of bicarbonate of soda or potash, citrate of potash, and tincture of nux vomica can be given before meals. The addition of vegetable bitters improves the appetite. Decoct. aloes co. dr.  $\frac{1}{2}$ -1 before meals may improve a poor appetite or reduce a voracious one. Give bismuth and carbonate of magnesia for diarrhoea; charcoal and salol for the tympanites and excess of mucus; small doses of calomel, if the stools are white. Maltine and mild alcoholic drinks are sometimes useful.



## CHAPTER XXV.

### DIARRHŒAL AFFECTIONS.

*Simple Diarrhœa—Infective Diarrhœa—Infantile Cholera—Ileo-Colitis and Colitis, catarrhal, ulcerative and membranous—Tuberculous Enteritis and Colitis.*

Diarrhœa is merely a symptom of many different affections. Some cases are functional, others of organic origin. They are divisible into two main groups: (1) Acute—mild, severe and choleraic; (2) Chronic—(a) functional; (b) organic.

On anatomical grounds the organic cases are divisible according to the main situation of the organic mischief, but many overlap. Thus, we speak of gastro-enteric catarrh and gastro-enteritis; enteric catarrh and enteritis; entero-colitis or ileo-colitis; and colitis. Or we may base the classification primarily on the grounds of etiology, and secondarily according to the morbid anatomy and severity, in the following manner:—

(1) *Simple or Non-Infective Diarrhœa.*

- (a) Mechanical, e.g., indigestible food, foreign bodies.
- (b) Dietetic, e.g., unsuitable food.
- (c) Nervous—due to excessive peristalsis, e.g., fright, fatigue, reflex irritation, atmospheric conditions.
- (d) Eliminative.

(2) *Infective, Inflammatory, or Febrile Diarrhœa*—toxic, fermental epidemic.

- (a) No serious anatomical change in the mucous membrane. This includes most febrile cases—mild, severe and choleraic, as well as toxic ones.
- (b) Serious inflammatory or ulcerative changes, secondary to the inflammation.

(3) *Symptomatic Diarrhœa.*

- (a) Due to gross anatomical specific changes, e.g., tuberculous, typhoidal and dysenteric.
- (b) Eliminative, e.g., uræmia and the crises of some febrile attacks, such as pneumonia and erysipelas.



The difference between simple and infective diarrhœa is that in the former there is neither microbial infection nor any special anatomical change in the digestive tract. According to this the toxic cases must be grouped with the simple diarrhœas. It must not be forgotten that a simple diarrhœa may become infective through secondary infection, and that in infective cases anatomical changes are not necessarily present. No strict line of demarcation can be drawn between the two types. Thus, it is obvious that no uniform system is quite satisfactory. The name sometimes indicates the cause of the attack, sometimes the clinical picture, and at others the locality of the pathological changes. According to the nomenclature of the Royal College of Physicians of London, the infective type should be certified as "*epidemic diarrhœa*," "*epidemic enteritis*," or "*zymotic diarrhœa*." The names are unsatisfactory, for enteritis is not always present nor do the cases always occur in epidemics. "*Summer diarrhœa*" is an unsuitable name, for it includes many cases occurring in hot weather which are of the simple variety and not infective, and the infective type may occur at other periods of the year.

The Registrar-General no longer recognises the names "*acute gastro-enteritis*" and "*gastro-enteric catarrh*," yet they are not inappropriate by reason of the clinical association of gastric and enteric symptoms, though there is no affection of the stomach, "*Entero-colitis*" and "*ileo-colitis*" indicate that both the small and the large intestine are involved. In many instances the colon escapes and in others it alone is affected. The name "*dysenteric diarrhœa*," given when there are mucus and blood in the stools, is unsuitable, for it is based merely on the character of the stools and has nothing to do with true dysentery. "*Choleraic diarrhœa*, *cholera nostras*, *cholera infantum* and *English Cholera*" are names appropriate for a particular type, with profuse watery diarrhœa and rapid collapse, but they are not true cholera. "*Febrile diarrhœa*" is not a perfect name, for though fever is almost invariable in the infective type, it may also be present in non-infective forms. "*Inflammatory diarrhœa*" is equally unsatisfactory for there is often no evidence of inflammation after death. "*Lientery*" or "*Lienteric Diarrhœa*" is an affection due to reflex peristalsis and is not true diarrhœa.

*Etiology*.—Infants are specially prone to diarrhœa for the stools are normally liquid or semi-solid. The food is liquid, peristalsis active, and the sphincter ani weak. Consequently the fæces are not long retained in the colon and diarrhœa may be a mere exaggeration of the normal conditions. Moreover, milk is a suitable nutrient medium for the growth of micro-organisms, and the deficiency of free hydrochloric acid in the stomach may enable them to escape destruction.

Unsuitable food sets up catarrhal conditions in the intestines and causes increased peristalsis. Rickets also predisposes to catarrh and the secretion of inefficient digestive juices. Heat affects the system generally



and impairs the digestion, for much less food is required. A deficient rainfall and long continued high external temperature predispose to epidemic outbreaks. These epidemics have been thought to depend upon the temperature of the soil, 4 feet below the surface, reaching 56° F. But the ordinary factors that come into play in hot weather are quite sufficient to account for the outbreaks. The bulk of the infective cases occur in the hottest months of the year, when the level of the ground water is low. A heavy rainfall reduces the number of cases and raises the height of the ground water, but it also reduces the temperature. The connection of diarrhœa with teething is discussed in the chapter on dentition. Age has an important bearing on mortality and the incidence of the attacks. Most cases occur between 6 and 18 months of age, and the mortality of infants under 1 year is appalling. This is due to the fact that additions to the diet are generally given about the age of 6 months.

The environment has an important influence on epidemic outbreaks and on mortality. Bad sanitation and hygienic surroundings lower the vitality of the child and exaggerate the effects and the dissemination of infection. Decomposing material is readily conveyed by flies to food or directly to the lips and mouth of the child. Unsuitable dwellings and overcrowding are injurious. The mortality among children living in "back to back" houses is higher than among those living in "through" houses. Among the labouring classes improvement in their dwellings is accompanied by a reduction in infantile mortality.

Anything which gives rise to catarrh in the intestines is a predisposing factor. Mere chill may act as an exciting cause of simple diarrhœa or predispose to infective attacks. Such a chill may be due to wet napkins, a prolonged hot bath, too cold water, short-coating, insufficient clothing, wet feet, etc. It is apt to follow sweating produced by rickets. The custom of dressing children, so that legs and arms are bare and the neck exposed, is much more likely to lower vital resistance and cause congestion of internal organs than to have a hardening effect upon the child. Intestinal parasites cause recurrent attacks by setting up reflex peristalsis or catarrh of the mucous membrane.

Under the age of 9 months, and to a less extent in older children, diarrhœa depends on the mode of feeding. Out of 1,000 fatal cases (Hope, 1887) only 30 of the infants had been entirely breast-fed. The mortality among infants brought up by hand was 22 times greater than among an equal number of those nursed or partly nursed. Artificial feeding, whether on cow's milk or proprietary foods, is the great cause of infantile mortality from diarrhœa. These observations have been confirmed by innumerable physicians. The attack is due to various causes connected with the diet. The food may be too rich in quality or too much in quantity, or both factors may be combined. It may be unsuitable in character to the age of the child. Many attacks are produced by malted or starchy foods



in the first few months of life ; and by indigestible articles of diet at all ages. In other cases the food is given too often or at irregular intervals. Deleterious changes may have taken place in it before ingestion. The milk may have gone sour or meat preparations may have "turned." Lactic acid is not a cause of diarrhœa, but in milk that has turned sour other organisms may have grown. The micro-organisms may produce toxins or ptomaines in the food before or after ingestion, or they may set up fermentative or proteolytic changes while in the intestinal tract.

In quite one-quarter of the cases other children in the same house are attacked. This means that they have been exposed to the same exciting cause, or that direct or indirect infection has occurred. Even the breast-fed infant may be infected by the mother's nipples, from lack of cleanliness or by organisms which have made their way up the ducts. Infants, too, are apt to suck the fingers, Job's comforters, teething rings, etc., and to put anything within their reach into the mouth. Nevertheless outbreaks of infective diarrhœa in the breast-fed are comparatively infrequent, and it is quite likely that infection is conveyed by flies or by neglect of cleanliness on the part of the mother. Epidemics have been traced to infection conveyed by milk, water or food at all ages. In institutions it may be conveyed from patient to patient by careless nurses. It is carried directly by infected dust, or indirectly through the infective dust being blown on to milk and other foods. On account of the prevalence of flies in hot weather and the rapid decomposition of organic matter in the room or in the neighbourhood, it is more than probable that the fly is the main carrier of infection.

There is a striking resemblance between infective diarrhœa and many outbreaks of *food poisoning*. They are clinically indistinguishable and may occur at any season. The simple form of food poisoning is due to toxins or ptomaines produced by the action of infective organisms in the food before it is swallowed. The symptoms come on rapidly, in a few hours after the ingestion of the poison, and are proportionate in severity to the size and virulence of the dose and the susceptibility of the individual. If the infective organism is present in the food and gets into the system, it may develop there and produce toxins. The symptoms do not arise as quickly but they are more prolonged and often more serious ; the case is really one of blood poisoning. Often the attack is due to toxins produced before ingestion and a secondary microbial blood infection subsequently. Here the acute initial attack may pass off and be followed, in the course of a few days, by a febrile condition due to the blood infection.

*Bacteriology.*—The meconium in the newborn is sterile, but by the second day the streptococcus acidilactici, *B. lactis aërogenes* and *B. coli* are abundant. Some attacks are apparently due to virulent forms of the *B. coli* which get into the milk, through fæcal contamination, and produce toxins. The most dangerous organisms are the *B. enteritidis* of Gärtner,



the *B. enteritidis sporogenes* of Klein, the *B. dysenteriæ* of Shiga, the paratyphoid and the paracolon bacilli, *proteus vulgaris*, and some streptococci.

*Pathology.*—In the infective cases the nervous symptoms, and to a great extent the systemic disturbance, are due to toxæmia. They are often out of all proportion to the diarrhœa and vomiting, which at first may be slight or absent. In other cases the constitutional symptoms are due to excessive loss of fluid. The vomiting is reflex in origin in most forms of simple diarrhœa; and in the infective type sometimes reflex and sometimes toxæmic. The different anatomical lesions are due to different poisons or to special proclivities in the individual. They are chiefly found in the cæcum and large intestine, that is in the places where fæcal material is longest retained. The local phenomena, diarrhœa and vomiting, are not often due to actual structural changes. To a great extent they are protective and the means of elimination of the toxins. Usually they are too profuse and have to be checked, and counteracted by the free administration of fluids. Œdema may be the result of renal inadequacy from the direct action of toxins on the renal epithelium. Broncho-pneumonia is generally due to secondary infection of the lungs. Sometimes in institutional epidemics it is part of a general septic infection of which the diarrhœa is also a symptom.

*Morbid Anatomy.*—The more acute the attack the less evidence of it is found after death. Often it is impossible, by examination of the cadaver, to ascertain the cause of death. There may be neither macroscopic nor microscopic changes. In more prolonged attacks there is a simple catarrhal inflammation of the mucous membrane, which varies in severity and distribution. The epithelium is denuded and the glandular cells are swollen and granular; the mucous membrane throughout the whole of the small intestine, more especially the lower half, or in patches of varying extent, is acutely inflamed. Sometimes the solitary follicles and Peyer's patches are swollen and stand out, much as in the early stages of typhoid fever. Sometimes this condition is so marked that bacteriological examination must be made for the typhoid bacillus in the spleen and mesenteric glands. Occasionally the mucous membrane of the stomach is also congested. In other cases, notably cholera infantum, the mucous membrane throughout the digestive tract is quite pallid and shows no signs of congestion. The contents of the intestines vary much in colour, resembling the stools passed during life, and are offensive; and there is a variable amount of gas. Coming to the colon we may find no definite anatomical change; congestion in patches or throughout; extensive proliferation or ulceration. In *Acute Catarrhal Colitis* the mucosa is swollen, covered with mucus, and exhibits patches of hæmorrhage into its substance. In one instance in a girl, aged 5 years, the swelling was so extreme that the colon formed a thick and rigid tube; this might be spoken of as *Acute Proliferative Colitis*. The thickening involved the whole wall, but especially affected the



mucosa. The child only lived 8 days. In a brother, aged 3 years, who also lived 8 days, acute inflammation was present with similar hæmorrhage and superficial abrasions, but no abnormal thickening. In prolonged cases of colitis the whole of the wall is involved, covered with a false membrane, and the solitary follicles are swollen and may ulcerate.

*Ulceration of the Colon* is of varied type. In severe catarrhal inflammation there is an irregular patchy erosion of the mucous membrane, due to superficial necrosis, which may extend in depth and area. Small circular ulcers either commence as an ulceration of the mucosa covering the lymph follicles, or the follicles undergo necrosis and form minute abscesses which discharge through the mucous membrane and leave a shallow ulcer.

*Follicular Colitis* occurs with or without ulceration of the follicles. In the milder type Peyer's patches are prominent and the follicles throughout the colon and, to a less extent, in the ileum are swollen and stand out above the surface as smooth rounded elevations, like minute peas; the overlying mucous membrane may or may not be swollen. In more advanced stages ulceration begins at the apex of the follicle and gradually extends deeper until the whole of the follicle is destroyed and extruded, leaving a sharply cut shallow pit-like ulcer. The colon may be honey-combed with such ulcers and some of them may coalesce into large ones, an inch or more in diameter. The ulceration extends down to the muscular coat, the striæ of which can sometimes be seen at the bottom of the larger ones. They do not cause perforation. In cases of long standing the ulcers may be pigmented.

In *Pseudo-membranous Colitis* the colon is mainly involved, and the lower 2 or 3 feet of the ileum generally affected. The walls of the gut are rigid and thickened. The mucous membrane is intensely engorged, red, swollen, and roughened in appearance or covered with a dirty greyish exudation of fibrin, which looks like membrane. The exudation consists of round cells, red corpuscles and micro-organisms embedded in a network of fibrin. It may infiltrate both the mucosa and submucosa, and the false membrane is then a compound of exudation and necrotic products. The intense engorgement is primary and results in a necrotic condition with patches of false membrane. Hæmorrhagic and petechial extravasations may occur. The other post mortem changes in the various forms of fatal diarrhœa are broncho-pneumonia, bronchitis and pulmonary collapse. The liver may exhibit fatty changes and collections of inflammatory cells round the portal canals. It has been suggested by Terrien that cirrhosis may start in this way. Cloudy swelling and desquamation of cells in the renal tubules have been noted, and are due to the fever or to the direct action of toxins. The brain may be congested or anæmic, and occasionally the cerebral sinuses are thrombosed.

**Simple Diarrhœa.**—In the breast-fed simple diarrhœa is due to colostrum in the first few days of life; to milk containing too much fat or too



many salts ; or changes in the milk due to illness, emotional disturbance, dietetic errors, etc., in the mother. One of the most frequent causes is too frequent nursing at irregular intervals. The milk contains too much protein, and the curds set up irritation of the mucosa, excessive peristalsis and diarrhœa. Chill is not an uncommon cause. The other factors are those already discussed in the general etiology of diarrhœa.

The stools may be green, the colour varying from patches of light green mixed with the fæces to a general watery green like spinach. The presence of curds shows that the milk contains an excess of undigested protein or fat. Fat curds are readily disintegrated by pressure. Yellow stools, more frequent and greater in quantity than natural, even thin and watery, are due to conditions of increased peristalsis and perhaps too liberal supply of digestible food. They may follow chill or may occur at the onset of infective diarrhœa. Profuse watery stools are also due to too rapid peristalsis ; they vary in colour from light brown to green, or may be clear, serous, like rice water. They are often dirty, offensive and putrid. Sometimes they are almost odourless or have a faint sickly smell. Slimy or mucoid stools from excess of mucus, are due to catarrh or more serious inflammation of the intestines, especially the large intestine. Bloody stools occur in acute inflammatory conditions, especially of the large intestine. It is unusual for any particular variety of stool to persist throughout the illness. They vary in character with the progress of the case, as well as with the anatomical changes.

In the bottle-fed and older children etiological factors are much more numerous than in the breast-fed, though they are chiefly dietetic in origin, especially in the simple cases. Attacks are liable to follow the ingestion of any indigestible kind of food, which sets up excessive peristalsis and perhaps some catarrh because of mechanical irritation.

*Symptoms.*—Simple diarrhœa is often ushered in or preceded by vomiting and a variable amount of colic. In the infant the knees are drawn up and the child utters an angry cry ; flatulence is commonly present. Vomiting, colic and diarrhœa are characteristic symptoms in all cases. Often in babies there is a variable degree of fever. At all ages, more especially in the very young, there may be considerable depression and even collapse ; the child being pale, the features pinched and drawn, eyes sunken, skin cold and clammy, and the temperature subnormal.

**Infective Diarrhœa.**—The symptoms are divisible into two groups ; the intestinal or gastro-enteric, and the nervous or toxæmic. The toxæmia produces the sudden onset, marked restlessness, incessant movement, sleeplessness, depression, prostration, cardiac collapse, vomiting and perhaps fever. The more acute the case the less is the fever. Some of the symptoms in the gastro-enteric form are due to thirst. The cases may be mild, severe or choleraic.



*Mild cases* simulate those of diarrhoea and vomiting due to unsuitable food. In babies the vomiting is slight and consists of curdled milk; colic may be present and the stools are unduly frequent, 5 or 6 per day, greenish in colour and containing curds. They may be sour and offensive and contain blood and mucus. In 2 or 3 days they are even much more frequent and more watery. The child is restless and irritable, no longer crawls or runs about, and looks ill. The face is pale and a little shrunken, the tongue furred, the pulse increased in frequency, and the muscles flabby. Appetite is impaired or lost and thirst is marked. Water alone may be taken readily and retained. The temperature ranges between 100° and 102° F. These patients recover in a few days; but often in a few hours may become very severely ill, passing on into the second type.

*Severe cases* are characterised by depression, collapse, and a temperature of 102-104° F. Cases often begin as mild attacks, but the fever increases instead of decreases, and all the symptoms are exaggerated. Vomiting is severe and often continuous. At first the vomiting consists of partially digested food, then of watery fluid and bile. The stools are very frequent more and more like dirty green or greenish water with brownish particles in them, or perhaps vivid green. Sometimes they contain mucus and blood. Often they are extremely offensive, and less frequently they are frothy. Tenesmus and prolapsus recti may occur. Restlessness is sometimes a marked feature from the commencement and throughout the illness. Thirst is often, but not always, extreme; it gives rise to restlessness, moaning, incessant movement of lips and tongue, and tossing of the head from side to side; even water is vomited. Gradually or rapidly the child becomes feeble, drowsy, and takes no notice. The face is drawn and pinched, expression anxious, cheeks pale and hollow, and the naso-labial folds exaggerated. The mouth is dry and the tongue raw, red and irritable, or shows patches of fur. In colitis the tongue may remain clean throughout. The eyes are half-open and staring, sunken, dull and lustreless. In late stages mucus collects on the insensitive corneæ and the pupils may be unequal. The fontanelle is sunken and pulsation barely palpable. The abdomen is swollen and tender, flaccid and clammy, or even retracted. Abdominal pain gives rise to acute cries, drawing up of the legs, and rigidity of the abdominal muscles. The skin loses its elasticity and is wrinkled. Œdema is not uncommon and general anasarca may occur. The urine is scanty, albuminous, and may contain hyaline casts. The pulse is small, frequent and running. At first breathing is shallow and increased in frequency, but towards the end it becomes slow, infrequent and sighing. Cyanosis is sometimes present. Finally, a comatose state results and death occurs from respiratory failure or is ushered in by convulsions. This comatose state is known as "*Hydrocephaloid*" or "*False Hydrocephalus*," for it resembles that of meningitis. The eyes are wide open and staring, the corneæ dull and insensitive, the pupils inactive to light and



perhaps unequal; but the fontanelle is deeply sunken and not bulging as in true hydrocephalus. The temperature may fall in the axilla, though remaining high in the rectum. Recovery is extremely rare from the hydrocephaloid state, and the mortality as a whole is high. Cases which recover usually end in 7-10 days, but the diarrhœa may persist 3-4 weeks or even become chronic.

*Choleraic* or *Fulminating Attacks* are sudden in onset, and soon exhibit grave toxic symptoms. They occur in the strong and healthy as readily as in the weak and delicate, and may terminate fatally in a few hours. At first the child is irritable, feeble, and prostrate. Soon it becomes dull and apathetic with pinched senile aspect, uttering feeble whining cries. Wasting is extraordinarily rapid. A strong child becomes a shrivelled wreck in a few hours. Thirst is extreme, and even water is almost immediately vomited. The stools are fæcal and copious at the onset. Soon they become watery, odourless and colourless, consisting of serous fluid as in true cholera; or they may be dirty brownish or greenish. Pain, abdominal tenderness, and tenesmus are usually absent. The temperature reaches 104-106° F. In some cases there is hyperpyrexia, and in others a subnormal temperature from collapse. Often the child is restless and sleepy, and becomes comatose towards the end. The attack may begin with or terminate in convulsions. Quite three-fourths of the cases prove fatal. The duration varies from a few hours to a few days. Recovery may be sudden and rapid, even in the apparently moribund. Death may occur before the onset of the diarrhœa.

**Colitis and Ileo-Colitis.**—Cases of this type are only differentiated from those of infective diarrhœa, by the anatomical changes found after death. In other words the disease must be in existence for a sufficient length of time to produce these changes. There is no definite line of demarcation, no essential difference in causation, and the one type may terminate in the other. On account of the severity of the anatomical changes and certain special peculiarities of the disease, an attempt must be made to define the chief diagnostic features.

Nothing need be added to what has been discussed under the general etiology of diarrhœa, except to insist upon the infective character of the affection. It may occur at any age, but is most common between 6 months and 2 years. It may occur at any period of the year, but is most common in the hot months. Sometimes it follows infectious diseases, such as measles, influenza and diphtheria. There is little doubt that it is contagious through infection from the discharges, and that different organisms may be responsible for different epidemics.

Its occurrence in epidemic form is rare in England. A well-defined outbreak came under my notice in 1900. All the cases occurred on one staircase in certain barracks. Four floors, with four rooms on each floor,



opened on to the staircase, and they were occupied by 13 women and 30 children. The outbreak began on the top floor, spread to the ground floor, and finally to the first floor. There were 12 inhabitants on each of these floors, and only 7 on the one which escaped. The outbreak began at the end of June and ceased at the end of August. It was probably due to an infective water supply from a cistern on the top floor. Six children were affected, and 4 of them died. Three of the women were attacked, but less severely, and recovered. Only one infant under 2 years was affected. This was a breast-fed baby, aged 3 months, who was infected either from the mother or another child. Two fatal cases, a brother and sister, aged 3 and 5 years respectively, ended in 8-9 days. The anatomical changes are described above.

The characteristic symptoms are an acute onset, with vomiting and diarrhœa, moderate fever, frequent pulse and respiration, great restlessness and depression. The stools are very numerous, small, offensive, green, and contain bright red blood, mucus and a little fæcal matter. In these cases there was no abdominal tenderness and no tenesmus. In the girl the abdomen was lax and empty; in the boy it was a little distended. Vomiting was frequent and persistent. The tongue was somewhat furred, and tended to become dry and brown in the centre, anorexia was marked, but there was no conspicuous thirst. The diarrhœa, vomiting and fever persisted, and death ensued from exhaustion due to the toxæmia. In favourable cases of this type, the vomiting subsides, the tongue remains clean throughout or soon becomes so, the character of the stools improves, and the signs of toxæmia are less profound. The blood disappears from the stools; next, the amount of mucus decreases, the fæcal matter increases, the stools become less frequent, and finally more solid. The offensive odour persists for some time, even after the stools are solid. As the diarrhœa ceases the fever subsides and the appetite returns. The anatomical lesions are those of acute catarrhal colitis, previously described. At the onset of these attacks the symptoms are probably put down to acute indigestion or food poisoning. Other symptoms are sometimes present, such as great thirst, abdominal pain, especially just before defæcation, tenesmus, tenderness in the course of the colon, and prolapsus recti. Although very frequent the stools are usually small in amount, perhaps not more than  $\frac{1}{2}$  oz. at a time. The blood is bright red, liquid or in streaks, and persists for a few days. The more the blood and mucus present, the worse is the case. The height of the temperature and the degree of toxæmia indicate the severity of the attack. In mild cases the fever may only last a few days and not reach more than 101° F. In a severe case it reaches 103° F., or more, and lasts for 7-10 days. The worst attacks prove fatal in a few days. If it is prolonged, it is liable to end in catarrhal ulceration and chronic diarrhœa. Ulceration is most prone to occur in the ill-nourished, in those badly treated at the onset, and from



too soon a return to ordinary diet. Even in the mildest cases convalescence is slow, a matter of some weeks or even months.

*Follicular Colitis* is the most common variety. It is rare under the age of 6 months and after 2 years; and is often found in fatal cases of infective diarrhœa in badly fed infants. The onset is usually that of infective diarrhœa of a moderately severe type, with vomiting and fever; or it may develop more gradually. Probably it is a primary catarrhal colitis with secondary extension to the lymph follicles, which become swollen and stand out above the level of the mucous membrane. Peyer's patches are also swollen. As the result of superficial necrosis of the mucosa covering the follicles, or necrosis of the follicles and secondary rupture, small ulcers are left.

The affection must be suspected in cases of infective diarrhœa in infancy in which the fever has persisted for over a week, and if the stools contain much mucus but no blood. In later stages, when ulceration has occurred, blood may be present in small quantities. The stools are not very frequent, but they vary in colour and are offensive. Their most characteristic feature is the presence of mucus. Round cells may be found on microscopic examination. Vomiting is not a marked feature, nor is the fever high, except perhaps at the onset. The appetite is lost, mouth dry, tongue dry and furred. Catarrhal stomatitis, thrush and simple ulcers are common. The abdomen is somewhat full, or occasionally relaxed and empty. Urine is scanty and loaded with urates. The skin is wrinkled and loses its elasticity, and emaciation is steadily progressive.

The course varies. There are frequently periods of remission followed by exacerbations which are readily induced by injudicious feeding. The ulcers never perforate, and their presence cannot be positively diagnosed, but must be suspected in prolonged cases and if blood is present. Relapses are common and complete recovery rare. Death takes place in a few weeks, usually 3-6, from exhaustion or from the marasmus secondary to the disease. It may be preceded by a hæmorrhagic petechial eruption on the abdomen and trunk.

*Pseudo-membranous Colitis* is a very severe variety affecting infants under 2 years. Clinically it is a grave type of catarrhal colitis with acute onset, vomiting, high fever, large liquid stools and prostration. Abdominal distension, tenderness, severe pain, tenesmus, prolapsus recti and considerable hæmorrhage are more constant and more marked than in the catarrhal variety. There is no essential difference in the stools, except that perhaps threads and patches of false membrane may be found. Prostration, delirium and coma may be so marked as to quite mask the abdominal symptoms. The diagnosis from catarrhal colitis is based upon the severity of the symptoms, the less continuous vomiting, the patches of false membrane in the stools, and in rare instances visible false membrane on the prolapsed mucous membrane of the rectum. It must be regarded as an



exceptionally severe form of catarrhal colitis with fibrinous exudation and superficial necrosis. It is almost invariably fatal in infants.

It is clearly quite impossible to make an exact diagnosis of the variety of colitis present, in many cases, though it can be assumed with a considerable degree of certainty. The changes in the colon mainly have been insisted on. But in about half the cases there is inflammation of the ileum as well. It chiefly affects the lower half, perhaps only 2 or 3 feet, as an acute catarrhal inflammation, general or patchy. Or it appears as a hyperplasia of the lymph follicles and Peyer's patches.

**Chronic Colitis** is divisible into chronic catarrhal, ulcerative, and membranous varieties. The chronic catarrhal form is sometimes called mucous, muco-membranous, pseudo-membranous or membranous; but the term membranous should be entirely dropped for this variety. It is a simple catarrhal inflammation in which the mucous membrane is swollen, red and thickened; and it is characterised by mucus in the stools. The mucus is discharged in glairy masses like slime, jelly or white of egg; sometimes they are so coherent as to float out in water and look like membrane, but they are readily disintegrated on stirring. The mucus is generally on the surface of the fæces and not intimately mixed therewith. It may be occasionally passed alone, in quantities as much as a wine-glassful at a time. Microscopically, there are found desquamated epithelial cells, leucocytes, blood cells, triple phosphates, and granular and fæcal debris.

This affection is quite frequent in children of all ages, though it is frequently overlooked. It is a sequel of an acute attack of ileo-colitis or of neglected constipation, through the irritation of the mucous membrane by scybala. Subjective symptoms are slight or absent. The child may appear perfectly well. Frequently there is a history of languor, disinclination to play, dark rings under the eyes, and perhaps a variable appetite. Often the appetite remains excellent throughout. The prognosis is good under simple measures of treatment.

*Ulcerative Colitis* is a sequel of the catarrhal variety, either acute or chronic, or due to specific infection as in tuberculosis, typhoid fever and dysentery. Stercoral ulcers result from obstruction and chronic constipation. The common variety of ulcerative colitis is the one in which the intestine is studded with many minute follicular ulcers. These cases have already been referred to under the name of follicular colitis. More extensive ulceration may be due to the aggregation of several follicular ulcers but, in spite of the frequency of follicular colitis, extensive ulceration is rare in infancy. Much more probably it is a sequel of the acute catarrhal variety or of chronic colitis secondary to constipation. The symptoms of the follicular variety are those of follicular colitis *plus* the passage of blood. Ulcerative colitis, similar in type to that of adults, is comparatively rare in children, and no fatal case has come under my observation. A



typical instance was that of a boy, 9 years old, under the charge of Dr. J. Smith, of Putney. Eight months previously he had an attack of diarrhœa and vomiting; 4 months later another and worse attack of the same nature from which he had not recovered. The general symptoms were slight febrile attacks with pain and vomiting, followed by the passage of loose, rather offensive stools, containing mucus and brightish red blood. Between the attacks the stools were somewhat the same in character, but less frequent. Nothing abnormal was found on physical examination, except slight pallor and a little tenderness along the transverse and descending colon. Another case of a milder type occurred in a girl, aged 6 years, who had been passing mucus and blood on and off for a year. Even under treatment the condition persisted for 8 or 9 months. The general health of these children and the appetite remain good, and for that reason it is difficult to get efficient dieting, nursing and treatment carried out. It is important in these cases to make a rectal examination, sometimes with the sigmoidoscope, in order to exclude local ulceration, foreign bodies, polypus, piles, angioma, papilloma and sarcoma.

*Membranous Colitis* is still more rare in children. It may be secondary to an acute attack; possibly primary and due to the pneumococcus or other organisms. In this condition there is considerable quantity of thick membrane composed of a fibrinous network, leucocytes, epithelial and red cells. In a case of a boy, 3 months old, with apparently simple catarrhal colitis, membrane was first found 7 months later, and for a year longer the lower bowel continued to form and discharge it in considerable quantities at intervals of a few weeks. Even 6 years later occasional shreds were passed, although the boy was in good health. This variety differs in no respect from the chronic catarrhal form, except in the character of the membrane, which must be distinguished from the muco-membranous casts of catarrhal colitis. It is almost certainly a variety of this, with more infiltration and destruction of the mucous membrane.

**Tuberculosis Enteritis and Colitis.**—Tuberculous ulceration may, or may not, give rise to diarrhœa, according to the situation and extent of the ulceration. Probably diarrhœa only ensues on secondary ileo-colitis, toxæmia or malnutrition. Most extensive ulceration may be found in the small or large intestine after death, although in life there has been no diarrhœa. An interesting case is that of a girl, aged 4 years, who had been ill for 11 months. For 6 months she had loss of appetite and wasting; then pain in the abdomen and diarrhœa began and continued in spite of treatment; for 2 weeks there had been blood in the stools. The child was very wasted, anæmic, and her liver was enlarged. The abdomen was a little distended, but at times was lax and empty, with no tenderness or local resistance. Vomiting was frequent and there was a little irregular fever. The stools were loose, very offensive, and contained small particles of clotted blood. All the symptoms persisted, vomiting became more



frequent, and the child died from asthenia in another 6 weeks. In the small intestine were multiple ulcers, many of them completely surrounding the gut; and the cæcum was extensively ulcerated. In the colon were 2 large irregular ulcers, one of which had caused a stricture with a lumen only the size of an ordinary penholder. There was no general peritonitis, but at the bases of the ulcers were miliary tubercles on the peritoneum, and some adhesive peritonitis matting together adjacent coils of intestine. Neither the mediastinal nor mesenteric glands were caseous. Caseous nodules in the upper lobe of the left lung made it almost certain that there was the primary focus of infection. Another case of very acute type, but ending in recovery, was that of a boy, aged 5 years, under Dr. Graham Morris, Wallington. Diarrhœa and irregular pyrexia, septic in type, often up to  $103^{\circ}$  F., had persisted for a month. The stools were frequent, loose and offensive, and contained much mucus. The abdomen was lax and empty, the spleen not enlarged, and a small lump was felt to the right of and below the umbilicus. The diagnosis lay between tuberculous enteritis and chronic ileo-colitis, probably the former, the lump being an enlarged mesenteric gland. One month later there were still frequent stools, 4 or 5 a day, with the occasional passage of blood-stained pus; and the temperature ranged between  $97^{\circ}$  and  $101^{\circ}$  F. This went on for another 2 months, and under an anæsthetic nothing but enlarged abdominal glands could be found. Under careful diet and treatment the boy completely recovered.

The usual symptoms in this variety of ulceration are wasting, diarrhœa, with offensive stools, which contain mucus and probably pus and blood, and variable pyrexia. Appetite is generally lost and reflex vomiting is not uncommon. Constipation may be present, although there is extensive ulceration. The disease is often limited to the small intestine and starts in the lymph follicles. It is almost always secondary to tuberculosis elsewhere, though it is possibly occasionally primary. The characteristics of the ulcers are the same as in adults, and the presence of tubercle bacilli in the stools is conclusive. The prognosis is not favourable, even under the most satisfactory conditions. Death results from exhaustion, peritonitis, and, rarely, perforation or hæmorrhage.

*Complications of Diarrhœa.*—Erythema and the excoriation of the skin about the buttocks, extending to the scrotum or vulva, are common results of the irritating discharges. Superficial ulceration is not infrequent. Pneumonia, lobular in type, is apt to occur in the infective varieties, being ushered in with fever, cough and frequent respiration. If the child is very ill, it may give rise to no special symptoms. The bacillus coli has been found in the lung in some of these cases. Pulmonary collapse is common in young children. Other complications are thrush, otitis media, thrombosis of cerebral or other vessels, and subcutaneous hæmorrhages. Petechial spots on the abdomen and back are generally seen in conditions in which there is profound marasmus, and are signs of an almost certainly fatal issue.



The child may recover from the attack but remain weak and delicate, with impaired powers of absorption and assimilation, and ultimately die from marasmus. Chronic diarrhœa results from atrophy of the gut, persistent enteritis or enteric catarrh, or chronic colitis. The infant remains ill, and does not gain weight, although food is taken ravenously and there is no vomiting. It steadily emaciates, the complexion is dull and earthy, the skin harsh and dry, and there may be profuse sweating. Irregular pyrexia is often present and continues for some weeks, and œdema of the extremities develops towards the end. The abdomen is distended and perhaps tender; colicky pains are not infrequent. Peristalsis may be visible through the thin parietes. The stools are abnormal and offensive, varying in different cases and during the progress of the same case. They may be greyish, pultaceous, and only a little increased in frequency; greenish or dirty brown, watery, frequent and offensive; or contain mucus and blood. On post mortem examination the mucous membrane of the small intestine may be much thinned and the glandular cells atrophied. In other cases catarrhal enteritis or follicular colitis is present.

*Diagnosis.*—Early stages of infective diarrhœa may be indistinguishable from those due to food. Subsequently the more than transient fever and the character of the stools are of value in diagnosis. Infective diarrhœa with prolonged fever has to be diagnosed from enteric fever. This is infrequent under 2 years of age and in isolated cases. Leucopenia, Widal's reaction, and an enlarged spleen are present. Intussusception, with passage of frequent small stools containing mucus and blood and frequent vomiting, may be mistaken for acute ileo-colitis; but it is rare for the opposite error to be made. The differential diagnosis of the seat of the morbid process is often impossible. There is no line of demarcation and many cases are compound in character. A simple infective diarrhœa, without gross anatomical changes, may develop into one or other of the different forms of ileo-colitis or colitis. If the catarrhal process is limited to the duodenum, there is rarely diarrhœa, and jaundice is almost invariable. Patches of isolated catarrh in the small intestines are indicated by the fæces being fairly solid and thoroughly mixed with lumps of mucus. If the affection is in the colon, the mucus covers or surrounds the fæces, unless they are liquid. Bright blood in the stools shows that colitis exists, and in tenesmus the rectum is involved.

*Prognosis* depends primarily upon the cause of the diarrhœa, and secondarily on the severity of the attack. A mild case may become rapidly acute and medical assistance is often sought too late. Simple diarrhœa is rarely fatal, except in marasmic infants and those suffering from other illness. The prognosis of infective diarrhœa depends upon the severity of the onset and the signs of toxæmia. Bad signs are continued high temperature, hyperpyrexia, or persistently subnormal temperature; continued vomiting and the passage of watery offensive stools; loss of elasticity of



the skin, œdema, scanty urine ; and the development of the hydrocephaloid state, cyanosis, broncho-pneumonia and collapse of the lungs. Favourable indications are the subsidence of bad signs, and the re-appearance of fœcal matter in the stools. A comatose state is rarely recovered from. The profuseness of the diarrhœa has not so much influence on the prognosis as would be expected. It is, however, a measure of the dose of the poison, and is a grave sign when there is much vomiting and consequent inability to take sufficient fluid to make up for that lost. Of the choleraic cases only about 1 in 4 recover. No case is hopeless, but the younger the child, the worse is the prognosis. Relapses are liable to occur and may be of greater severity.

If there are gross anatomical changes, the outlook depends upon the severity of the attack ; the health and strength of the patient ; the environment and the weather ; and on the chances of a chronic inflammatory state persisting. In symptomatic diarrhœa the probability of recovery depends on the severity and upon the associated disease. An acute eliminative diarrhœa in uræmia, or a critical discharge in pneumonia, erysipelas and other fevers, may prove fatal.

*Treatment.*—The preventive treatment consists in the education of the mother, or attendants, in general attention to the child's health ; regulation of the diet, the avoidance of stale food ; good hygiene and ventilation ; cool climates or sea-air, if the weather is hot, and bathing in cold, not tepid, water. Whenever a child passes a watery green stool, it requires medical treatment. Once the attack has commenced we have to replace the drain of fluid, destroy or get rid of infective germs, and keep the child alive until the attack is over. In the adult gastric juice contains hydrochloric acid in sufficient quantity to prevent development of putrefactive organisms, but in the infant hydrochloric acid is deficient in quantity. Many of the lower animals secrete a gastric juice which contains no ferment, but is very rich in acid, and they are able to eat infected food with impunity. Antiseptics by the mouth may have the very reverse effect to that intended. Small doses of some of them interfere with the action or the activity of the enzymes of the digestive tract ; and large doses render them inert. Carbohydrate food is nutritive and antiseptic. It is unsuitable as a medium for the growth of proteolytic bacteria, and it lessens the albuminous putrefaction in the intestines by producing lactic and other organic acids which destroy or inhibit the growth of many dangerous organisms.

*Simple Diarrhœa.*—First get rid of any irritant matter by means of a dose of castor oil, calomel or grey powder, sodium or magnesium sulphate ; and, secondarily, attend to the diet. In the breast-fed infant, it is usually sufficient to regulate the nursing according to the principles of breast-feeding. If the child is over 6 months of age, weaning may be advisable. In the bottle-fed it may be sufficient to regulate the feeds. Give a smaller quantity of a weaker food at longer intervals, or dilute the milk with a weak



solution of arrowroot or rice water. Often it is enough to stop the administration of some proprietary food to which the attack is due. It may be necessary to peptonise the milk for a few days, or even to get a wet nurse. Should the attack be due to febrile conditions, as in teething, the quality and quantity of the food must be reduced. Attention must be paid to the milk supply, the mode of preparation and administration of each feed, the temperature of the food, and the cleanliness of all the apparatus. Hot foods increase the motility of the stomach, and iced water diminishes it. Both are liable to set up reflex intestinal peristalsis, so it is advisable that all food be given lukewarm.

In older children the diet must be limited for at least 24 hours. It should consist of equal parts of boiled milk and thin arrowroot or corn-flour gruel. Complete starvation is often advisable, or milk should be entirely omitted. In every case the patient must be kept warm, and put to bed if the attack is severe.

After the preliminary purge drugs are rarely required, but the return to the ordinary diet must be gradual. Give bread and milk, tea and toast, boiled egg, bread and butter, cornflour and ground rice puddings, fish and mashed potato, and so on. Drugs are often necessary and useful. For green stools in infants, the addition of sod. bicarb. or sod. citrat., grs. 1-5 to each feed, is frequently efficacious. If the stools contain curds, small doses of castor oil m. 3-5 with mucilage and caraway water, 4 times a day, frequently cause constipation in 2 or 3 days. For catarrhal or inflammatory states of the alimentary tract, especially if mucus and blood are present in the stools, give bismuth in large quantities. Even a drachm of the carbonate or subnitrate may be given as an initial dose. More usually frequent doses of grs. 5-10 are sufficient. A suitable mixture consists of bismuth carb., glycerin., pulv. acaciæ, aa dr. 1., aq. cinnam. to 2 oz.; 1 dr. being given every 2-4 hours for infants of 6 months. In these cases it is often advisable to give in addition grey powder, gr.  $\frac{1}{4}$ - $\frac{1}{2}$ , bis or ter in die. To any of the above mixtures tr. opii. m.  $\frac{1}{4}$ - $\frac{1}{2}$  may be added, if there is any pain or if the diarrhœa is uncontrolled. It is generally best given alone in water, and repeated according to the effect on the child. It is particularly useful in cases of profuse diarrhœa and excessive peristalsis, in which the stools are shot out as from a pump. Older children may be given bismuth grs. 10-20, or pulv. cretæ. aromat., with or without opium, grs. 10-20, for a few days. Astringents are very rarely required. As a rule they are merely of temporary benefit and are followed by recrudescence signs. They may be useful in prolonged chronic cases.

The treatment of infective diarrhœa consists of starvation, elimination, warmth, stimulation, the administration of much fluid, good nursing and cleanliness. Subsequently measures for the reduction of inflammation are needed. The principles of treatment are the same in all cases, requiring modification with the severity of the attack. Except in very bad attacks,



begin with an initial dose of castor oil or calomel ; the latter if there is much vomiting. Even in the worst attacks frequent doses of calomel, gr.  $\frac{1}{20}$  -  $\frac{1}{10}$  hourly, are beneficial. For babies over 9 months of age, sod. sulphat. grs. 10, every  $\frac{1}{2}$ -1 hour for 4-6 doses, is better than castor oil or calomel. It does not cause vomiting, and it flushes out the bowel, eliminates toxins, and reduces the temperature. If there is much fever, it is very useful ; but it is contra-indicated by low temperature, great debility and sunken fontanelle. It can be given even when there is blood in the stools, if used early. If there is much vomiting, the stomach can be washed out with warm alkaline solution. This gets rid of food, mucus, fermenting products and toxins, and permits the introduction of a dose of castor oil before taking out the tube.

*Diet.*—In acute cases milk must be discontinued for 12-48 hours, and nothing given by mouth except plain hot water every hour, or in quantities of dr. 1 every  $\frac{1}{4}$  hour. It is of little use giving food which will be at once vomited. Water, even if vomited, washes out the stomach, relieves thirst, is diuretic and eliminative. Hot weak tea, cold water, toast water, and rice or barley water may be given if preferred. Albuminous foods, even albumin water, should be avoided, if the stools are putrid or contain mucus or blood, and when the toxæmic symptoms are marked. The attack is most frequently due to organisms which grow best in albuminous foods. In these cases cereal decoctions are much more beneficial. The value of carbohydrate food in the prevention of intestinal putrefaction has been already discussed (pp. 272-3). On the other hand carbohydrates are contra-indicated when there are flatus and abdominal distension, acid and frothy stools, and no constitutional symptoms. Unsweetened condensed milk, weak peptonised milk and albumin water are suitable in such attacks.

Meat broths are generally permissible, for they are little more than solutions of extractives, almost devoid of protein and carbohydrate. They are stimulants rather than food, and can be given in most acute cases after 24 hours, or before, if they can be retained. Sometimes they help to maintain the diarrhœa. Fresh meat juice, dr. 1 every  $\frac{1}{4}$  hour, can sometimes be retained when everything else is vomited. In hot weather it is constantly full of microbes and may prove more dangerous than the disease. On the whole there is little benefit, and perhaps much risk in the use of meat preparations. The proprietary foods containing albumoses and peptones actually cause diarrhœa. The jellies are generally pleasant and harmless. After the acute stage or as soon as food can be retained, give whey, white wine whey, unsweetened condensed milk, cream and water, or diluted peptonised milk. Then try Benger's Food, milk and barley water, arrow-root gruel, and gradually return to the ordinary diet. On account of the great liability to relapse and to chronic inflammatory mischief in infective cases, the diet must only be increased slowly.



*Elimination* is assisted by subcutaneous injections of normal saline solution, small enemata of saline, and irrigation of the colon according to the methods described under rectal therapeutics. The temperature of the fluid for colon irrigation should be 60-70° F., if there is much fever; 100° F. in cases of medium severity with much mucus; and 110° F., as a stimulant, if the temperature is subnormal or the patient weak. Irrigation in acute colitis washes away mucus, germs and irritating particles of undigested food, but cannot reach the deeper layers of the inflamed mucous membrane.

Subcutaneous injections are also useful in collapse. From 50-150 c.c. of normal saline solution should be injected every 6 hours. It is introduced quite slowly into the loose cellular tissue of the abdomen, axilla, groins or thighs. Marfan recommends an injection of 1-4 drs. every 4 hours of a mixture of caffeine citrate 0.75 gms., sod. chlorid. 2.5 drs., sterilised water 300 c.c.

*Stimulants* are best given as small doses of brandy or whiskey, m. 3-10, in hot water every 1-3 hours. In choleraic cases and collapse it can be given freely by mouth or rectum. The next most valuable stimulant is strychnia gr.  $\frac{1}{400}$  hourly and given subcutaneously. Or we can make use of sal volatile, m. 3-5 in water, or subcutaneously in extreme stupor; sulphuric ether m. 5-15 by mouth or sub cutem; musk gr.  $\frac{1}{2}$ -2 every 15-30 minutes until grs. 5-10 have been taken; camphor dissolved in olive oil, 1 in 30, and given sub cutem, or by mouth mixed with glycerine in doses of gr.  $\frac{1}{4}$ -2, or spirits of camphor m. 5-10, every  $\frac{1}{2}$  hour by the mouth and less often subcutaneously.

The child must be kept very quiet, the mouth cleaned with weak alkaline solution, and the room well ventilated. The nurse must carefully disinfect her hands after attending to the child, wash before she prepares the meals, and disinfect all napkins.

*Bathing*.—Infective cases must be kept very clean and, if there is much fever, should be sponged after the usual methods (p. 75). Hot baths and mustard baths are given as stimulants in collapse and prostration. A hot bath, for 5-10 minutes night and morning and given very gently, allays restlessness and to some extent relieves thirst and aids elimination. A tepid bath at 80° F., with the addition of a handful of sea-salt in 4 gals. of water, is useful in convalescence and as a preventive measure in hot weather. It may be followed by a cold douche at 60° F., if the child is old enough.

*Drug Treatment*.—Purgatives, antiseptics, sedatives and astringents are all used. Acacia is added for the suspension of soluble drugs and as a sedative for the gastric and intestinal mucosa. Glycerine is better than syrup as a sweetening agent, as it does not ferment in hot weather. At the onset small doses of calomel, gr.  $\frac{1}{24}$  -  $\frac{1}{8}$ , may be combined with Dover's powder, gr.  $\frac{1}{4}$ , and given every 2 or 3 hours for 5 or 6 doses. After the acute stage it should be given every 4-6 hours, with the addition of bismuth carb.



grs. 10. Bismuth compounds are valuable sedatives, mildly astringent. They are said to be antiseptic, to stop the evolution of gas, to be alterative, and to exert a tonic action on the stomach and bowels. The subcarbonate is the lightest, is most free from grit, and most pleasant to take. A suitable mixture for a baby 6 months of age, consists of the subcarbonate, glycerine, acacia aa dr. 1., aq. carui to 2 oz., given in doses of dr. 1 every 1-3 hours. The salicylate, subnitrate or oxide can be used in similar doses. To the above 2-oz. mixture there may be added acid carbol. gr. 1-2, paregoric m. 15-30, and sodii bicarb. dr. 1. Bismuth preparations should be preceded by a mild purge, kept cool, well shaken before using, and given frequently. They are chiefly of value after the acute stage. The bismuth is said to be more active when converted into a sulphide, and for this purpose the addition of sulph. præcip. gr. 1 to bism. subnit. grs. 10 is sufficient.

For excessive foetor give the sulphocarbates of soda, lime and zinc, aa gr.  $\frac{1}{6}$ - $\frac{1}{2}$ ; or phenol m. 1, tr. iodi. m. 1, glyc. m. 5., aqua ad. dr. 1; or tr. iodi. m. 1, glyc. ac. carbol. m. 1, aqua ad. dr. 1.

Innumerable antiseptics have been used with varying success. Sodium salicylate is highly recommended by Jacobi, but in my experience it is of little value. Salol gr. 1 every 1-2 hours, alone or combined with bismuth and given in sugar-coated tabloids, is of more use. It breaks up into salicylic acid and phenol in the intestines, and should not be given for more than 3 days. Phenol, combined with bismuth and aromatic chalk powder, is also valuable. Of the naphthalin group benzo-naphthol and beta-naphthol give the best results, but are often useless. They are given in doses of gr. 1 for 24 hours, for each month of life under 1 year of age, and in doses of grs. 5-10 combined with equal quantity of sugar 3 times a day for older children. The stools become odourless, the fever less, and the general condition improves. They can be combined with bismuth preparation, and small doses of Dover's powder. Naphthalin, naphthol, phenocoll, and betol (b-naphthol salicylate) have a somewhat similar effect. Creosote prevents fermentation and acts favourably in tuberculous ulceration. Styrcol grs. 5-10 is also useful in this type of ulceration. It passes through the stomach unchanged, and is converted into guaiacol and cinnamic acid. If intestinal antiseptics are given the most reliable are carbolic acid, calomel, b-naphthol, salol, iodine, iodoform, chlorine, creosote and guaiacol. Turpentine is useful for hæmorrhage and prevents meteorism.

Astringents are injurious, until the alimentary tract is clear of poisonous products. They are comparatively of little value and rarely advisable, especially in infants. Tannin irritates the stomach, and sets up pain and vomiting. It can be given, without producing such effect, in the form of tannigen, alone or combined with bismuth and other drugs; or as tannalbin, a powder containing about 50 per cent. of tannic acid, insoluble in the mouth and stomach, and resolved in the intestine into tannin and albumin. It is given in doses of grs. 2-10, and is useful for chronic and tuberculous



diarrhœa. Other astringents are silver nitrate, gr.  $\frac{1}{8}$ - $\frac{1}{6}$ , given in water with acid. nitrici dil. m. 1, 3 times a day; zinc oxide grs. 1-5 with bismuth grs. 5-20 every 2 hours; and drugs such as logwood, coto, kino and catechu. A useful powder consists of calomel gr.  $\frac{1}{8}$ - $\frac{1}{4}$ , pulv. kino co. gr.  $\frac{1}{2}$ , pulv. ipecac. co. gr.  $\frac{1}{2}$ , 1-3 times daily, in chronic cases with restlessness.

*Opium* and its preparations must not be given in early stages, for they interfere with elimination and may paralyse the intestines. Opium is more efficacious than codeia. It allays pain and restlessness, reduces shock, and promotes sleep. It must not be given if the tongue is furred, if there is much vomiting, or in the presence of collapse. In small doses it is beneficial if the stools are frequent, watery and accompanied by straining; if the tongue is clean and the stools are offensive on account of excessive peristalsis; and if blood and mucus persist. It may be used in late and chronic stages, and is also valuable in reflex or lenteric diarrhœa. In bad cases it is best given alone, in the required dose, and repeated according to circumstances. In less severe ones it may be added in minute doses to bismuth or castor oil mixtures. The best preparations are tr. camph. co. m. 3-10, tr. opii. m.  $\frac{1}{4}$ - $\frac{1}{2}$ , and pulv. ipecac. co. gr.  $\frac{1}{10}$ - $\frac{1}{3}$ , every 2-3 hours. As an enema tr. opii. m. 2-5 in 1 oz. of starch can be given to an infant over 6 months of age, for it produces topical effects and is more slowly absorbed. Morphine may be given subcutaneously in doses of gr.  $\frac{1}{100}$  under 6 months of age, and gr.  $\frac{1}{30}$  over 1 year of age. Opiates must be given cautiously, the pupils watched, and the child never awakened for its dose. With these precautions no evil effects result from small doses.

*Serum treatment* has not proved of much value up to the present, except in cases of dysentery and those due to Shiga's or Flexner's bacillus.

In *chronic colitis* treatment is carried out on the same lines. Mild cases recover on simple dietetic measures, warmth, bismuth, and attention to the bowels. Foods containing indigestible residues must be avoided. If due to simple chronic constipation a liberal fruit diet or a modified cellulose diet may be beneficial. A saline purge, such as Apenta water, is given every morning. In more severe attacks the child must be kept in bed and fed on peptonised milk, lactose and eggs. Iodoform pills or creosote mixtures are given if the stools are offensive; mild purgatives or enemata for constipation; large doses of salicylate of bismuth and aromatic chalk powder in acute diarrhœa, or some of the drugs above recommended. A diet of curdled milk or butter milk is sometimes of value for intestinal infection, proteolytic organisms being crowded out or destroyed by the lactic acid bacilli. If there is ulceration, the bowel can be irrigated with solutions of common salt or boric acid, tannin or tannic acid 1 per cent., or silver nitrate gr.  $\frac{1}{4}$ -1 to the ounce. The silver nitrate injection is preceded by irrigation of the colon, and from 5-20 oz. are injected, according to the age of the child (p. 87). It can be repeated every 3 days for 3 or 4 times and is then discontinued for an interval of 10-14 days. If it causes much



pain, a small dose of opium may be given. The other silver compounds are not nearly as efficacious. Should this fail, appendicostomy will enable the bowel to be washed out daily with alkaline, antiseptic or astringent lotions. The results of appendicostomy have been fairly satisfactory in dysentery in adults, but less so in ulcerative colitis. One great drawback to it is that it leads to secondary contraction of the colon, rendering it impossible to close the opening. Even in these cases the patient is not certainly cured, and may have more or less hæmorrhage from time to time.

*Symptomatic treatment* must be adopted in many cases with a view to the relief of the most urgent symptoms. For hyperpyrexia the cold pack with ice to the head is the best remedy, though too vigorous for most children. In mild cases ice suppositories, cold rectal injections, tepid or cold sponging, and cradling may be sufficient. In the worst ones the cold bath may be essential. The temperature must be taken in the rectum, or it may not be realised that the blue, cold, apparently collapsed child is in a state of hyperpyrexia.

Vomiting must be stopped, for it is exhausting and neither aperients nor stimulants can be retained. The usual methods recommended for the treatment of vomiting (p. 252) must be adopted. Collapse is treated by alcohol and strychnia; mustard applications and heat in various forms, e.g. rectal irrigations; and the subcutaneous injection of salines and stimulants. It is probably due to the action of the poison on the splanchnic area, and is very analogous to shock. The subcutaneous injection of m. 3 of a 10 per cent. solution of chloral hydrate; atropin. sulph. gr.  $\frac{1}{500}$  -  $\frac{1}{1000}$  hourly, as an antidote to the toxæmia, until the pupils dilate; or glonoin gr.  $\frac{1}{200}$ ; or atropine gr.  $\frac{1}{1000}$ , morphine gr.  $\frac{1}{100}$  hourly are all recommended.

For cerebral symptoms give a hot bath, or apply hot bottles to the feet and ice to the head. Tenesmus is relieved by a warm injection of starch and opium, oil or gelatine; the local application to the anus of cocaine ointment 5 per cent. or ichthyol 1 in 8; and the administration of Dover's powder.

Every case must be treated on its merits; bearing in mind that some attacks of diarrhœa are simple and harmless, others infective and virulent, and a few chronic and persistent. The bulk of the cases are due to errors in diet, and on a suitable diet the patient may rapidly recover. There are no hard-and-fast rules of treatment appropriate for rich and poor, for the intelligent and the stupid, for the well-nourished and the marasmic, in the heat of a tropical climate and the cold of more northern regions. With care and patience investigate every case, ascertain its causation and the nature of the pathological process. Attend to the minutest detail in symptoms and treatment, giving the most careful directions to the attendant, for it is on these minutiae that success in the treatment, of infantile diarrhœa in particular, especially depends.



## CHAPTER XXVI.

### CONSTIPATION.

*Simple Constipation—Dilatation of the Colon—Hirschsprung's Disease.*

**Constipation** means infrequent action of the bowels or the passage of hard, dry fæces at an age when they should be semi-solid or almost liquid. The tendency to regular daily evacuations is inherited and varies in different families. The habit can be cultivated by careful training. It is advisable that the bowels should act once or twice a day in the first year of life, as otherwise the consistence of the fæces becomes hard. For older children daily evacuation is advantageous, though not absolutely necessary.

*Constipation in Infants.*—Complete obstipation in the newborn depends on atresia or stenosis of some part of the alimentary tract. Later on it may be due to hypertrophic stenosis of the pylorus, dilatation of the colon, the pain and spasm caused by anal fissure, or some of the mechanical forms of obstruction more common in older children. The infant is normally predisposed to constipation, because the sigmoid flexure is tortuous and up to 2 years of age is relatively much longer than in later life. Apart from these causes it is generally functional or dietetic in origin. It occurs in both the breast-fed and the bottle-fed, more frequently in the latter. The dietetic causes include lack of food, profuse vomiting, defective quality of food and unsuitable composition. If the milk is too watery, it may be fully digested and assimilated, but the stools are small and infrequent, the urine excessive in amount, and the child wastes. If the quantity is deficient, constipation is accompanied by scanty secretion of urine. Deficiency of fat in the milk is a common cause; so, too, sterilised milk and starchy foods, unless they set up fermentation. The functional causes are those dependent upon deficient intestinal secretion, weak muscles, and defective nerve impulses. Deficient peristalsis may depend on atony of the gut, impaired reflex excitability or a mental defect, such as the cretinoid state. It may be produced by some of the popular remedies for colic and the various soothing mixtures. The intestinal secretions are deficient in fever and malnutrition, and the stools become dry, friable, and chalk-like.

*Symptoms.*—The baby may be brought for treatment because of colic, constant screaming, flatulence, restlessness, insomnia, and muscular



twitchings. Wasting is often present in cases due to an insufficient food supply, as well as in others in which the food does not appear defective. Toxic symptoms are rare because of the simple character of the food. Anorexia, flatulent eructations, vomiting after each feed, fever, and even convulsions may occur. Vomiting, associated with constipation, generally indicates that the food is excessive in quantity or too rich in quality. On physical examination there may be nothing abnormal except a little abdominal distension. In severe and prolonged cases the abdomen becomes tense, drum-like and tender. The increasing tympanites pushes up the liver, and the splenic dulness becomes obliterated. Secondary effects, such as umbilical or inguinal hernia, prolapse of the rectum, hæmorrhage, piles, and dilatation of the colon may be found. The stools are scanty and dry. Hard, whitish, scybalous masses are voided with much pain and straining. Wasting is consequent on imperfect digestion, insufficient absorption of nutriment, and the effects of colic and restlessness.

*In older children* the condition may be due to persistence of the infantile habit, atony of the intestines, deficient innervation, faulty diet, or defective intestinal secretions. The intestinal atony may be hereditary, muscular or nervous in origin. Frequently it is acquired as a result of recurrent constipation, inattention to the calls of nature, or the continued use of large enemata and cathartic drugs. Sometimes it depends on mechanical obstruction, such as intussusception, hernia, peritoneal adhesions or inflammation, and stenosis of the gut. Deficient innervation leads to deficient peristalsis and may be hereditary or secondary to nervous affections, such as imbecility and meningitis, or to atrophy of the gut aided by deficient secretion. The dietetic causes are chiefly an insufficient supply of fluid, fat or indigestible residue; the excessive or too early use of starchy foods (this may cause diarrhœa); astringent wines, tea, and vegetables containing tannin. Bolting food and imperfect mastication are additional factors. Most of these causes, and fever also, lead to deficiency of intestinal secretions. Constipation occurs in rickets and other constitutional diseases which give rise to anæmia and general debility.

*Symptoms* are not uncommon, except in trivial cases, and are generally toxæmic in character. The child is dull, languid, easily tired, depressed and irritable; of a sallow, muddy, yellowish complexion, with dark rings under the eyes; and suffers from headache, bad or capricious appetite, flatulence, nausea, and disturbed sleep. The tongue is coated, breath foul, abdomen often distended, abdominal pain common, and the child wastes. The stools are hard, lumpy, dark coloured, and covered with mucus. A scanty evacuation takes place daily or much less frequently. Hard masses may be felt in the descending colon, and the irritation gives rise to rectal discomfort and useless straining efforts, catarrh, and the exudation of mucus in which thread worms readily live. Intermittent attacks of fever are sometimes entirely due to constipation. The chief secondary results



are proctitis, anal fissure, prolapsus recti and piles. A less frequent but more important sequel is dilatation of the colon.

*Treatment in Early Infancy.*—First relieve the retention of fæces, and then try to cure the habit. In the simplest cases in breast-fed infants attend to the diet and habits of the mother, with the object of reducing the percentage of protein and increasing the percentage of fat in the milk and the total quantity. In order to accomplish this, prescribe a liberal nitrogenous diet, a moderate amount of alcohol, tonics, plenty of exercise, and prolong the intervals between each nursing. The administration of a teaspoonful of cream in warm water, twice a day before nursing, is sometimes a sufficient remedy. For a bottle-fed child give a mixture containing a high proportion of fat and sugar, and at a suitable age give malted food or oatmeal. Brown sugar and treacle are somewhat laxative. Water should be given freely in hot weather. The habit of evacuation at the same hour daily must be taught. At first it may be necessary to stimulate the action of the bowels by the introduction of the oiled tip of the finger, the nozzle of a syringe or a soap suppository. If these simple measures are insufficient, give a lump of manna the size of a piece of sugar once or twice a day; or in each bottle-feed sulphur gr. 1, or sodium phosphate or citrate grs. 5-10; or one dose daily of fluid magnesia, cream of magnesia, or mag. carb. levis grs. 2-5. If the stools are chalky and white, tr. podophyllin m. 1-2 may be given 2 or 3 times daily. Peristalsis is encouraged by abdominal massage along the colon, in a circle round the umbilicus, for 5-10 minutes 3 times a day. The skin is rubbed first with oil to prevent excoriation. After the first year of life the tips of the fingers may be applied and no oil used. Treat the colon as if you were trying to empty a long sausage. Begin over the sigmoid, then direct the movements from the splenic flexure to the sigmoid, and next from the hepatic flexure onwards, and finally from the cæcum. Soap suppositories consist of gr. 1 of soap to m. 5 of oil of theobroma, and are comparatively harmless. Small glycerine suppositories, gluten suppositories, and a simple cone of oiled paper are of similar value. They should be well oiled before insertion, and used once or twice a day. To obtain immediate relief inject glycerine dr.  $\frac{1}{2}$ -1, in water oz.  $\frac{1}{2}$ . Other useful injections are oz.  $\frac{1}{2}$ -1 of cold water, soap and water, salt solution or olive oil. Medicated suppositories containing aloin, ext. belladonnæ, ext. nuc. vomicæ aa gr.  $\frac{1}{24}$ - $\frac{1}{12}$ , can be used after the age of 2 years, but are of little more value than the simple suppositories. The prolonged use of suppositories or injections, whether of soap or glycerine, may set up catarrh of the rectum.

Internal medication is often needed for chronic and severe cases. The doses should be given regularly, 1-3 times daily, in sufficient quantity to keep the bowels open without purging, and continued for weeks or months, being gradually omitted. Manna, aloes, sulphate of soda; the syrups of senna, rhubarb and figs; hyd. c. cret., alone or with pulv. rhei co.; and



fluid magnesia or cream of magnesia, are generally used. At 6 months of age a mixture containing tr. aloes or tr. aloes et myrrh. m. 3-5, tr. belladonnæ m. 1, tr. nuc. vomicæ m.  $\frac{1}{2}$ , syr. zingiberis m. 15, and water to a drachm, 3-4 times a day, is generally efficacious. It can be given more frequently and the dose of aloes increased. It is reduced as soon as a loose stool is passed. Sometimes maltine, alone or with cod liver oil, is sufficient. Drastic purges should be avoided. The usual dose of castor oil is followed by recurrence of constipation. For hard fæcal masses give an injection of warm olive oil, dr. 1-4, followed in a few hours by an injection of soap and water or salt solution, oz. 1-4. At 2 years of age an injection of ol. terebinth dr. 1, ol. olivæ drs. 2, and the yolk of 1 egg, well mixed with 4 oz. of warm water can be used. Injections must be given gently and slowly, and stopped at once if pain is produced. Mechanical aids, such as a bent hairpin, a scoop or a teaspoon, are rarely needed.

*Treatment in Older Children.*—In mild cases rely on hygienic measures, diet, habit cultivation, posture at stool, massage, and general and abdominal exercises. The diet should contain plenty of fatty foods and waste matter. If it fails, it does harm by causing dyspepsia and increasing the amount of residue to be got rid of. A liberal supply of water is necessary. On waking give fruit, such as an orange or apple, and half a tumblerful of hot water; and a full tumbler at night. Reduce the amount of cow's milk, which alone is a frequent cause of constipation, and give a liberal supply of fruit, porridge, wholemeal stale bread, and green vegetables. Olive oil, cod-liver oil, petroleum emulsion, terrol and paroleine are useful, and act mainly as lubricants. The squatting posture should be adopted at stool. If the ordinary closet is used, the seat should be small and the feet raised on a high stool. Massage should be carried out at bedtime with the child on a hard mattress, the head and shoulders raised, the legs flexed and a little rotated outwards. It should be done for 5-10 minutes, while standing on the left side of the patient and using the tips of the fingers in the manner above described. It can be applied by the child, using a rubber ball filled with 2 or 3 lbs. of No. 10 shot and rolling it over the abdomen for 10 minutes night and morning. It can be supplemented by an injection of olive oil, oz.  $\frac{1}{2}$ , and by galvanism or faradism of the abdomen.

Of all drugs the various preparations of aloes and cascara are the most valuable. They exert a stimulating and tonic effect on the lower bowel. The ordinary aloes pill and the mixture of aloes, belladonna, nux vomica and ginger, recommended for babies but in larger doses, are most satisfactory. Sodium sulphate can be added if necessary. Nux vomica or strychnia is generally essential in order to increase muscular activity and peristalsis. Cascara is best given in the form of the aromatic syrup or as cascara and malt. If there is evidence of deficient hepatic activity and defective intestinal secretion, add tr. podophyllin, or give small doses of calomel or grey powder and rhubarb. Other mild preparations are the



syrups of senna, rhubarb and figs; confections of sulphur and senna; sulphur lozenges; tamarind pulp, tamar indiens, and laxative fruit pastilles containing senna. The recent synthetic drugs, such as purgen or phenolphthalein, laxoin, and exodin in doses of gr.  $\frac{1}{2}$ -2 are tasteless, efficacious, and harmless, and act in much the same way as salines. Continued enemata are liable to cause ballooning and irritation of the rectum, and render it atonic. Glycerine is unsuitable for prolonged use; it is too stimulating and makes the rectum tolerant of lesser stimuli. If enemata are used, give olive oil oz.  $\frac{1}{2}$ , soap and water or plain cold water 2-3 oz.

The most useful purges for children are castor oil, rhubarb, sulphate of magnesia or jalap, calomel, grey powder or blue pill, and aloes. Liquorice powder, calcined magnesia with or without rhubarb, and mineral waters, such as Apenta, are also valuable. Castor oil is the most harmless. Rhubarb, especially in the form of Gregory powder, is particularly useful if there are stomachic conditions. Salines are preferable, if a watery evacuation is desired; mercurials, if the function of the liver is at fault; and aloes, if the defect is in the muscular activity of the lower bowel. In purgative doses they do not cure constipation. If they are given for this purpose, they must be given systematically, and the dose reduced as soon as a single loose stool is passed. For severe cases aloes and sodium sulphate combined are given in increasing doses until 2 liquid stools are passed daily. The full dose is continued for 2 or 3 weeks, and then reduced slowly. The aloes or aloes and iron pill is used in a similar way.

To relieve chronic accumulation give a full dose of castor oil and a warm olive oil enema, followed by soap and water enema in a few hours time. If this fail, give extract belladonna gr.  $\frac{1}{4}$  every 2 hours until the pupils are widely dilated. Complete impaction is treated by calomel gr. 1-5, followed by castor oil and large injections of thin warm gruel containing ol. ric. oz. 1, ol. terebinth oz.  $\frac{1}{2}$ ; or pure olive oil; or ox gall and water p.a. This may be combined with the belladonna treatment and mechanical assistance.

**Dilatation of the Colon.**—The persistence of chronic constipation, fæcal obstruction, foreign bodies, and stricture may cause considerable dilatation of the colon. In the so-called idiopathic variety, or *Hirschsprung's Disease*, there is no evidence of any obstruction. In other instances there may be marked narrowing, to the size of a lead pencil, of the rectum and colon (Atkin); the sigmoid flexure and rectum (Treves); the ascending and transverse colon (Dodd). These cases of congenital narrowing of a portion of the gut must be distinguished from those of idiopathic dilatation. The symptoms date from birth or shortly after, and may be delayed for a few years if the narrowing is not sufficient to cause definite constipation. The ordinary sequence is constipation, abdominal distension and intestinal obstruction.

Hirschsprung (1887) reported 2 cases of the idiopathic variety.



Tuffier (1907) collected 88 cases, of which one was in a 7 months foetus, 21 in the first year of life, and 61 over 19 years of age. These are cases which are not due to faulty habits, chronic constipation, or the abuse of drugs or enemata.

*The Etiology* of the condition is uncertain. There is evidence that dilatation may depend on the nerve and blood supply. Thus "ballooning" of the rectum is due to defect in the muscles, and is often associated with conditions affecting its innervation and blood supply. Excessive tympanites of the small intestine results from thrombosis of the superior mesenteric vein, and has been produced by ligature of the main mesenteric vein in animals. Probably there is a neuro-muscular defect of congenital origin, leading to inertia of a portion of the colon. Hawkins reported a case in a baby, aged 6 months, whose grandfather had been under his care for a similar condition and had died from it at 40 years of age.

*Morbid Anatomy.*—The sigmoid flexure is divisible into the iliac colon, occupying the left iliac fossa; and the pelvic colon, which extends from the inner edge of the psoas into the pelvis, there forms a loop and turns back along the posterior wall of the pelvis to the third piece of the sacrum, where it joins the rectum. The loop has a mesentery and is entirely free. In the child it varies much in length, is relatively longer than in the adult, and lies in the abdomen. At the junction of the pelvic colon with the rectum is an increased amount of circular muscle fibres, forming a kind of sphincter. Possibly a similar kind of sphincter is present at the junction of the colon and sigmoid, a colo-sigmoid pylorus, and the fæces are stopped and moulded in the sigmoid. Dilatation almost invariably begins in the lower 3 ins. of the pelvic colon, not in the rectum. This suggests a predisposing anatomical factor. Sometimes it is extreme, at the time of death, and there is no hypertrophy. More commonly there is secondary hypertrophy of the circular muscle fibres and the muscularis mucosæ. The wall may reach  $\frac{1}{4}$  in. in thickness, the bowel forming a rigid tube. Other changes are probably secondary. Concetti has noted cases in which areas of hypertrophy of the large intestine alternated with atrophic portions.

No definite organic stricture is found. The anus is normal; and anal spasm can be excluded, for the rectum is not funnel-shaped, though sometimes decidedly roomy. The pelvic colon usually shows funnel-shaped dilatation, but the lower 2 or 3 ins. may be unaffected. The enlargement is rapid, but not abrupt, somewhat resembling the sloping neck of a bottle, reaching a diameter up to 8 ins. The loop shape of the pelvic colon is generally preserved. It rises out of the pelvis, and the butt end of the loop may reach the right costal margin. If the mesentery is short, it may become stomach-shaped. The rest of the colon may be normal, or more or less dilated for a variable distance, rarely as far as the cæcum. The small intestine is collapsed and empty. The contents of the colon are nearly



always semi-solid, greenish-yellow, somewhat like wet sand, inoffensive and deficient in faecal smell; sometimes there are scybala. There is almost invariably much gas and this must be regarded as the dilating force.

*Symptoms.*—Constipation is present from birth, or an early age, and may alternate with diarrhoea. There may be no spontaneous evacuation of meconium. Usually constipation is incomplete and flatus is passed. Sometimes the bowels only act once in 3-6 weeks, and complete obstipation of 2 or 3 months' duration has been reported. Gradually the abdomen enlarges and is often asymmetrical, with prominence in the left iliac region. It is surprisingly flaccid and rarely dull, the resonance being due to the formation of gas in the colon, which becomes enormously dilated, tympanitic and distended. The pelvic loop, extending up to the umbilicus or costal margin, may be visible through the thin parietes; and peristalsis may be obvious and excessive, though it is absent in great distension. The abdomen becomes hugely distended, forming the "balloon belly," but still remains flaccid and doughy. The splenic and liver dulness are obliterated. The superficial veins are dilated, and the skin may be oedematous. The lower part of the thorax is so distended that the costal margin forms almost a straight line. The mechanical pressure produces shortness of breath, displacement of the heart upward, palpitations, intermittent pulse, oedema of the legs and scrotum, and possibly albuminuria. The dyspnoea may be so great as to cause lividity and inability to move. Vomiting and pain are rare; diarrhoea may result from passive overflow, secondary catarrh and ulceration, with possibly the passage of bloody stools. Indicanuria may be present. In the terminal stages there may be abdominal pain, hiccough, constant vomiting, increased micturition, cyanosis, coldness, collapse and syncopal attacks, or pyrexia due to toxæmia. The general health remains remarkably good for a long time, and the final stage begins with dyspeptic symptoms and loss of weight. Death is due to chronic obstruction and stercoræmia, acute obstruction, or ulceration, perforation and peritonitis.

*Prognosis.*—Many cases die in infancy. Thus, a girl, 8 weeks old, under my care, died from collapse due to the perforation of one of three stercoral ulcers 8 ins. from the anus. She had been costive since birth. The large intestine was greatly distended from the cæcum to the sigmoid, especially in the transverse colon, and its contents were like wet sand. There were neither scybala nor stenosis. Few cases live to be over 40 years of age. The affection is rarely thought serious until purgatives, and then enemata fail to give relief. Attacks of obstruction occur at intervals. Untreated cases steadily get worse and develop failure of compensation, stagnation, obstruction, toxæmia, and the effects of mechanical pressure. Death may be sudden and unexpected. Ulceration is found in about one-third of the cases after death.

*Treatment* is unsatisfactory, unless the dilatation is due to chronic constipation, or depends on more or less stenosis of the gut. Constricted gut



has been removed with satisfactory results. In the idiopathic cases acute obstruction must be treated by immediate colotomy, unless the bowels can be quickly emptied by the persistent use of castor oil and olive oil enemata, and mechanical means. In the chronic cases similar methods are adopted, in conjunction with careful dieting, small and concentrated meals, saline purgatives, strychnia, tight bandaging, abdominal massage and faradism of the gut. Massage is dangerous in bad cases. Surgical measures are generally advisable for idiopathic cases. Colotomy may prolong life but is not curative. Excision of the dilated portion of the colon has given good results, but the part that is left is liable to undergo subsequent dilatation. Anastomosis of the iliac and pelvic colon and fixation of the dilated pelvic loop, with subsequent excision of the loop, has also been tried. On the whole it is justifiable to explore and empty the bowel by manual pressure, before trying further measures. If this is unsuccessful, appendicostomy and systematic lavage of the colon may be tried. So far I am unaware of any case having been treated by this method, but it seems justifiable, seeing that it leads to contraction of the colon in cases of colitis.



## CHAPTER XXVII.

### INTESTINAL PARASITES.

*Tapeworms—Roundworms—Threadworms—Trichinæ—Myiasis.*

**Tapeworms** (Cestoda) are flat compound worms with no mouth or alimentary canal, multiplying by gemmation from a pyriform head or nurse. Each segment, or proglottis, contains ova, which are set free when the segment is discharged from the human body. The ova are then swallowed and pass an intermediate stage of existence in another host, in which the embryo comes to rest in some part of the body, being transformed into a bladder-like structure, called a *cysticercus*, *bladder worm*, or *measle*. It is swallowed in this form by the child. In the stomach the wall of the cysticercus is dissolved and the head, or scolex, is set free. This attaches itself to the mucous membrane of the intestine and develops into the mature worm. It attains full maturity in about 3 months, and may live 20-30 years or longer.

Tapeworms occur at any age, but are rare under 5 years. It is exceptional for more than one to be present in the same child. The *Tænia Solium* or pork worm, "*ver solitaire*," is acquired by eating insufficiently cooked sausages, pork, etc. It attains the length of 6-10 feet. The head is the size of that of a pin, and provided with 4 suckers, a rostellum and a circlet of hooklets. The fully developed segments are almost square, a little longer than broad.

The *Tænia Mediocanellata* vel *Saginata*, or beef worm, is acquired by eating insufficiently cooked infected beef. There may be more than one. It attains the length of 12-20 feet. The head is provided with 4 suckers, but no rostellum or hooklets. The segments are larger than those of *T. solium*, and longer than they are wide. It is the most common tapeworm in children.

The *Bothriocephalus Latus* is rare in this country, and acquired from fresh-water fish. It attains the length of 24-60 feet. The head is small, ovoid, with two lateral suckers. The segments are much wider than long.

The *Tænia Elliptica* vel *Cucumerina* (dipylidium caninum) passes its intermediate existence in lice and fleas of cats and dogs. It is the common tapeworm found in cats and dogs, and rare in human beings. It has been found at the age of 6 weeks, and possibly many tapeworms in infants are of this kind. Its length is 6-12 ins. The head has a rostellum,



circlet of hooklets and sucking discs. The segments are long, narrow and somewhat ovoid.

The *Tænia Nana*, or dwarf tapeworm, is found in Central and South Europe, America and Egypt. It consists of 150-200 segments, which are small, oval, reddish-white, like minute gherkins. The head has a rostellum, circlet of hooklets and sucking discs. The *tænia flava punctata* has been occasionally found.

The *Tænia Echinococcus* lives in the dog and such-like animals. The ova are swallowed in watercress, etc., and the embryos develop and pass into the tissues, especially the liver, and form hydatids.

*Symptoms* may be absent; or may be due to the worm, catarrh of the intestine, or drugs given for treatment. The growth of the worm at the expense of the host withdraws food supplies, notably albumin, and leads to malnutrition. Mechanically it may give rise to obstruction, or by migration may set up myositis, meningitis, liver abscess or asphyxia. Such results are rare, except from hydatids. Some of the symptoms are due to reflex irritation, and others are toxæmic in origin. The common abdominal symptoms are variable appetite, nausea, vomiting, discomfort or colic, tympanites, and constipation or diarrhœa. Rarely a tapeworm gets into the stomach and is vomited. Constitutionally, it may produce irritability, uncertain temper, restless nights and malnutrition. Erythematous rashes may be due to toxæmia or secondary digestive disturbance. The nervous symptoms include cough, itching of the nose and anus, giddiness, tinnitus, pavor nocturnus, hallucinations, hysterical symptoms, habit spasm, chorea, convulsions and epilepsy. It is difficult to believe that some of these affections are more than coincident; the symptoms having been perhaps exaggerated by intestinal catarrh and toxæmia. Severe cough and serious loss of weight may be due to worms. The cough is dry, noisy, incessant or occurring in fits of alarming intensity, and lasting for hours but without râles or fever. In a boy, 33 months old, the symptoms were strongly suggestive of meningitis, and cleared up on the evacuation of a tapeworm. The diagnosis depends on the appearance of segments in the stools, and from these the variety of worm can be determined.

The *prognosis* is good as regards life, but uncertain as regards the evacuation of the worm. The drawback to drugs is that the most efficacious ones are unpleasant to take. They are liable to affect the stomach, may not be retained, and may cause severe enteritis, toxic symptoms and death. No such drugs should be given, unless the diagnosis is certain. Their use is contra-indicated by early age of the patient, recent abdominal disease, and uncompensated heart lesions.

*Treatment*.—For *tænia elliptica* give calomel and santonin. For the common tapeworm the extract of male fern is the best drug, but it may have to be given in large doses. Care must be taken that the drug is soon evacuated, for even small doses may give rise to toxic symptoms and an



erythematous rash. In the case of a child of 4 years or more of age, it is advisable to have the bowels opened freely by mag. sulph. dr. 1 and tr. jalap m. 15, or such a dose as may be necessary, for 4 or 5 days before giving the male fern. No food should be given after 1 p.m., except possibly a little weak tea at bedtime. The child is kept in bed during the course of treatment on the following day. At 7 a.m., it is given a mixture of ext. filic. maris. liq., ext. glycyrrhizæ liq., syr. zingib., aa. dr. 1, in 1-1½ oz. of water. If it is not retained a second full or half dose is given in an hour's time. Two hours later castor oil oz. ½, or a full dose of the mag. sulph and jalap mixture, which has been found to act quickly and efficaciously, is given. Tincture of jalap can be given with the castor oil. If the bowels do not act in 3 hours, an enema should be given. For the next day or two the diet must be a light one. If the head of the worm is not found, careful watch must be kept for the reappearance of segments in the stools, and the treatment repeated in about 3 months, when the worm is well developed. It dies if separation takes place shortly behind the head. Male fern can be given in doses of m. 15 of the liquid extract or oleoresin in capsules every quarter of an hour for 4 doses, instead of the mixture recommended above; the other treatment being the same. The oil of filmaron is more pleasant to take and has been recently recommended. In the case of dogs it is given in castor oil, without previous starvation, and an efficient purge given an hour later. But it must not be given on successive days.

It is hardly necessary to mention other drugs, seeing that they are not nearly as efficacious, but sometimes it is advisable to adopt less severe treatment. Black oxide of copper has been recommended by Hager (1888) and Sass (1898). A mixture of black oxide of copper gms. 5, prepared chalk gm. 1, mag. carb. gm. 1, tragacanth gms. 10, glycerine gms. 5, and white sugar gms. 40, is made up with water into 50 pastilles. Two or three are given daily for a fortnight. Nothing acid must be taken and rest in bed is not required. Debris appears in 2 or 3 days.

Fresh pomegranate bark, macerated in an equal quantity of hot water and boiled down to half its bulk, is given in doses of drs. 6-12 on two successive mornings, and followed by a purgative. Other remedies are pumpkin seeds in doses of oz. 1, pounded up with sugar and honey into a paste; cucumerin, a concentrated extract of the seeds which tastes like beef juice; kousso, an infusion of 1 dr. for half an hour in 3 oz. of boiling water, and given in 3 doses at intervals of 2 hours, preceded by castor oil the night before; pelleterine; and turpentine.

**Roundworms.**—The *Ascaris Lumbricoides* is acquired through the ingestion of the ova or embryos in drinking water or contaminated vegetables. An intermediate host is unnecessary. These worms are very common in children from 1-10 years of age, especially in Cornwall, and almost universal in the natives of India and the West Indies. They are more frequent in girls than boys, usually multiple, and inhabit the small



intestine. Although generally harmless, they are dangerous on account of their migratory habits. Thus, they have passed into the trachea and caused fatal asphyxia, atelectasis or pneumonia; into the pancreatic duct, setting up interstitial pancreatitis; into the bile duct, appendix, and through a typhoid ulcer; into the Eustachian tube, through the external ear or nose; and through an umbilical fistula. It is even said that the worm may perforate a healthy intestine. They have also given rise to intussusception, intestinal obstruction, and peritonitis.

The *symptoms* are mainly those of indigestion, viz., anorexia, variable appetite, nausea, vomiting, abdominal pain, tympanites, diarrhœa and offensive stools. The pain may be vague and about the navel; occasionally acute, causing moaning and crying. In other cases the symptoms are limited to headache, constipation, wasting, pallor, debility and syncopal attacks. The chief nervous symptoms are picking the nose, grinding the teeth, restlessness, sleeplessness, somniloquence and pavor. Occasionally there are found irregular pulse, unequal pupils, contracted fields of vision, transient squint, hemiplegia, aphasia, vertigo, hallucinations, tetany, chorea, laryngeal spasm, meningitic symptoms, jaundice, enuresis, palpitations, crying fits, hysterical symptoms, unconsciousness, convulsions, and fever.

Worm fever, "*fièvre vermineuse*," or "*lumbricose à forme typhoïde*," may closely resemble typhoid fever or the cerebral type of meningitis. The temperature rises to 104° F., with malaise, anorexia, headache, thirst, and occasionally rigors, delirium, and epileptiform convulsions. The symptoms are due to reflex irritation and toxins, excreted by the worm or from decomposition of dead worms in the duct. The presence of normal stools enables many other causes of the child's illness to be excluded. Usually a large number of worms are present in these cases, rarely one only.

These worms are readily evacuated by giving santonin on an empty stomach, preceded by a laxative and followed by castor oil or calomel. A suitable powder consists of santonin gr.  $\frac{1}{6}$ , calomel gr.  $\frac{1}{6}$ , pulv. scam. co. gr.  $\frac{1}{2}$ , for each year of age, and given at bedtime for 3 or 4 nights. Santonin should never be given in large doses. The urine becomes very yellow and turns red on the addition of alkali. An excessive dose produces headache, vertigo, vomiting, dilated pupils, yellow vision and convulsions. It may be fatal. Goosefoot oil, *ol. chenopodii anthelmin-tici æthereum*, is said to be almost a specific for roundworms. It is given in drop doses with sugar and water, or as an emulsion. It is not difficult to take, acts promptly, and has no unpleasant effects.

**Threadworms.**—The *Oxyuris Vermicularis* chiefly inhabits the rectum and lower part of the colon; is common in the cæcum and appendix; and has even been seen in the stomach and mouth. No intermediate host is needed, and apparently they breed in the intestines. They are found in large numbers, embedded in the mucus resulting from the catarrh



to which they give rise, and lodged in folds of the swollen mucosa. Catarrhal states predispose to their development. It is doubtful whether they can live in a healthy mucous membrane.

They give rise to itching of the anus and genitals, soon after going to bed, due to migration of the worms from the rectum. This may induce local sores, enuresis, balanitis, vulvitis and masturbation. Constipation is usually present, but catarrhal colitis, proctitis and diarrhoea are not infrequent. Possibly they set up appendicitis. The nervous symptoms include itching of the nose, restlessness, irritability, nervous twitchings, convulsions and chorea. Eosinophilia is present in less than half the cases of threadworms and tapeworms, and often absent in roundworms. It may occur in children without apparent cause. A diagnosis is made by inspection of the anal region and stools. Wash out the rectum with cold water, and examine the fluid against a dark background. Seek for eggs under the microscope.

*Treatment* is directed to curing the catarrh and evacuating the worms. Reduce the starches, sweets, vegetables and fruits in the diet; and give a daily dose of hyd. cum. cret. gr. 1, pulv. rhei. grs. 5, sod. bicarb. grs. 5. Small doses of perchloride of iron with a saline laxative; alkalies with gentian and rhubarb; gentian and quassia; sulphur lozenges; garlic; naphthalin gr.  $\frac{1}{2}$ -5 t.d.s., omitting fat from the diet; and petroleum emulsion are also useful. Powders of santonin, calomel, and scammony are as valuable as in its treatment of roundworms.

Rectal injections of common salt, 5 per cent. solution, and infusions of quassia, garlic, asafoetida or weak perchloride of iron are given in order to get rid of the worms in the lower part of the bowel. The salt solution is the simplest, cheapest, most harmless and, in my experience, is efficacious as well. It washes away the mucus in which the parasite lives. Strict cleanliness is necessary, for patients re-infect themselves by scratching and getting the ova under the nails. The nails must be kept short; and gloves and tights worn, if necessary. The anus must be washed after each stool, and freely anointed with ung. hydrarg. nit., diluted if necessary, every night for 6 weeks, to destroy the parasite as it emerges or prevent its emergence. If these measures are insufficient, all drinking water must be boiled, for it is sometimes a source of infection. Uncooked fruit and vegetables should be washed before being eaten.

The *Trichocephalus Dispar* is very common in the cæcum, but rarely gives rise to ill effects. It is an occasional cause of enteritis, appendicitis, reflex colic and nervous symptoms. The ova are found on microscopical examination of the stools. The treatment consists in attention to the intestinal tract, and drugs such as thymol, benzine and male fern.

The *Anchylostoma* or *Dochmius Duodenalis* is a dangerous inhabitant of the alimentary tract, but unknown in this country in children. It causes



great anæmia, from the abstraction of blood by the parasite and bleeding from the local punctures. It is treated by thymol and male fern.

**Trichinosis.**—Trichinæ are rare in this country. The embryos are swallowed in imperfectly cooked measly pork. In the stomach they are set free and, passing into the intestine, multiply with great rapidity and cause gastro-enteritis, sometimes profuse diarrhœa, and even superficial ulceration in the upper part of the small intestine. The young worms then penetrate the intestinal walls, and reaching the muscles set up myositis with local tenderness and swelling. There they become encapsuled and finally calcified. The constitutional symptoms of the intestinal stage and the muscular invasion may be severe and simulate typhoid fever. The disease is often fatal, if the number of trichinæ swallowed is at all large. Calomel, anthelmintics, and intestinal antiseptics, followed by castor oil, are indicated in the treatment of the intestinal stage, but it is rarely diagnosed and generally is assumed to be simple ileo-colitis. The muscular stage is treated on general principles.

**Myiasis.**—Accidental parasitism of dipterous larvæ, the maggots of flies, is occasionally seen. Ambrose Paré (1582) reported a case in which a maggot was passed per urethram, and similar cases are on record. Maggots are not infrequent in cutaneous wounds, the nose and the external auditory meatus. They are readily cured by blowing in calomel, and then syringing with warm boric acid lotion; by warm oil, carbolic acid lotion 2 per cent., chloroform vapour or tobacco juice. In rare instances subcutaneous swellings have developed, broken down, and extruded maggots. These are due to some fly depositing its eggs under the skin.

*Myiasis interna*, or gastro-intestinal myiasis, is occasionally seen. Usually the eggs of a fly, or the larvæ, are swallowed, and perish in the intestinal tract. If they effect a lodgment, they may give rise to enteritis and colitis from mechanical irritation or toxins. Vomiting, fits of screaming, and attacks of colic have also been noted. The larvæ may be vomited or passed in the stools. In many supposed cases they are due to subsequent infection of the excreta. Calomel, thymol and naphthalin are the most useful drugs.

**Protozoa.**—The chief protozoa are the amoeba coli, cercomomas intestinalis, trichomomas intestinalis, megastomum entericum, and balantidium coli. They are usually taken as encysted forms in water, raw fruit, vegetables and salad. For treatment give a slightly constipating diet, cure the intestinal catarrh, and prescribe drugs, such as calomel, thymol, tannalbin, and quinine tannate. Irrigate the bowel with quinine or thymol solution 0·2 per cent. or tannin 0·5 per cent. strength.



## CHAPTER XXVIII.

### INTESTINAL OBSTRUCTION.

*Malformations—Volvulus—Intussusception—Hernia—Congenital Hydrocele.*

Intestinal obstruction occasionally follows abdominal operations, as the result of adhesions. Sometimes it is caused by the adhesions of tuberculous peritonitis, but there is the danger of mistaking the constipation and vomiting at the onset of meningitis for that due to obstruction. Anatomical defects, except Meckel's diverticulum, are rare. The mesentery has been found attached to the spinal column only, and the colon behind instead of in front of the small intestine (Thursfield's case). Obstruction, from abnormalities in connection with Meckel's diverticulum, causes localised meteorism and bulging above or below the umbilicus, tenderness to the right of the umbilicus, and early fæcal vomiting. The navel may be indrawn or scarred. Sometimes there is a past history of minor attacks.

**Congenital Intestinal Atresia and Stenosis.**—Intestinal atresia is much more common than stenosis, and, apart from the rectum, is decidedly infrequent. Silbermann (1882) collected 24 cases of duodenal atresia and 30 of atresia, but only 3 of stenosis in the jejuno-ileum. Gärtner's (1883) statistics of the site of atresia were duodenum 16, jejuno-ileum 20, large intestine 2. Schlegel's statistics (quoted by Braun, 1902) were duodenum 29, jejuno-ileum 54, large intestine 6. Atresia is most common in the lower portion of the jejuno-ileum, generally in the ileum near the cæcum, usually with a cord connecting the two ends. It is rare in the colon and, when present, generally in the first part.

*The Duodenum.*—Cordes (1901) collected 56 cases and added one of his own. Of these, 48 were cases of atresia and 9 stenosis. The close relation to the bile duct is important. The defect may be above or below, and rarely on a level with the entrance of the duct. Bland Sutton states that the bile duct almost always enters the distal extremity of an imperforate duodenum. The duodenum may be completely occluded, ending in a blind pouch or converted into an impervious cord. D'Arcy Power has reported a case in which the obstruction was due to a complete transverse septum, composed of mucous membrane and circular muscle fibres, in the second part of the duodenum. The child lived 5 days.

*The Jejunum-ileum* may contain single or multiple occlusions. As a



rule there is complete atresia, the ends of the gut ending blindly but united by a band continuous with the mesentery. A narrow lumen may be found sometimes in the band, and occasionally muscular tissue and intestinal glands. The band may be absent, and the ends of the gut in close apposition or widely separated and in different parts of the abdomen. Sometimes the occlusion is due to a diaphragm, with a central perforation, formed by mucous membrane, and the intestine is surrounded externally by a furrow. Congenital multiple occlusions of the small intestine have been repeatedly described. Stenosis and atresia may co-exist in the same patient. In stenosis the lumen may be so small as to prevent the passage of the contents of the bowel. The gut is distended above, often much thickened, and collapsed below the stenosis. In the cases of multiple occlusions normal meconium has been found in the gut, even in portions which are completely shut off.

*The Colon* is rarely occluded. In intestinal atresia the small and large intestine below the obstruction are very contracted and empty, the bowel being about the thickness of a lead pencil. An absence of continuity between the small and large intestine has been reported (Souter), and other rare developmental anomalies, e.g., absence of the cæcum and ascending colon (Clogg); absence of the whole colon, except the cæcum, and sigmoid (Nash); absence of the descending colon and sigmoid (Westwood Fyfe). In Nash's case, a girl of 5 years, a foreign body was successfully removed from the small intestine by operation. The large intestine was represented by a short tube, passing from the cæcum to the brim of the pelvis on the left side and continued as the rectum.

*Etiology.*—Usually there are no other malformations, but there are anomalies in position and length of the gut. There is no common cause. Most cases are due to developmental errors in early foetal life, for it is unusual to find bile in the distal segment. Possibly it is a failure of the lumen to become patent. Duodenal atresia is associated with the development of the liver and pancreas. The development of the liver, as an outgrowth from the duodenum, and of the pancreas may be irregular. Other malformations have been described in connection with the development of the liver, viz., diverticula of the duodenum near the orifice of the bile duct, anomalies in the bile and pancreatic ducts, and an accessory liver or pancreas. Intestinal atresia is associated with Meckel's diverticulum, which may cause atresia by producing strangulation. This will not explain the cases in which the occlusions are multiple. Cases have been ascribed to intussusception, volvulus, constriction in the umbilical ring, compression by tumours, and obliterating arteritis. Braum and Chiari found the remains of intussusceptions. There is no satisfactory evidence in favour of foetal peritonitis, though traces of it have been found. The occlusion takes place too early in foetal life to render it probable. Nor is there evidence of embolism or thrombosis of mesenteric vessels, nor of intestinal ulceration.



In one instance (Pearce Gould, 1882) the obstruction was due to plugs of inspissated mucus in the lower ileum and colon. The plugs contained no bile, so must have been formed before the third month of foetal life.

*Symptoms* depend on the situation of the obstruction. The higher up it is in the intestinal tract, the earlier the vomiting begins. In Souter's case it began as soon as the head was born. Usually it starts on the first or second day. If the occlusion is above the entrance of the bile duct, no bile is found in the vomit, and the case may be mistaken for one of stenosis of the pylorus; but the symptoms are earlier in onset and more urgent. If the obstruction is low down, the vomit contains food given; bloody matter from the gastric mucosa, the result of straining; and perhaps meconium. These cases also show abdominal distension, visible and violent peristalsis, and gurgling. Practically nothing is passed per anum, though the anus and rectum are normal. If the obstruction is above the entrance of the bile duct, meconium is passed. The symptoms are those of starvation—pinched and drawn face, dry tongue, subnormal temperature, feeble pulse, scanty or suppressed urine, rapid wasting, collapse, and both tonic and clonic spasms. Emaciation and exhaustion terminate the scene in 4-7 days, rarely longer. Abdominal distension may be so great as to cause dyspnoea and cyanosis. Treatment has so far proved useless.

**Volvulus.**—A congenital twist of the third part of the duodenum was found by J. F. Gordon (1906) in a girl of 6 years, who died from asthenia after 15 weeks of intermittent vomiting. Volvulus of the ordinary type is rare in children. It affects the sigmoid, is very uncommon in the small intestine, and is said not to occur in normal bowel. Complete volvulus includes the entire small intestine, cæcum and ascending colon. It is due to a congenital arrest of development, and is more common in boys than girls. Volvulus causes severe abdominal pain, early and persistent vomiting, constipation, and other signs as in adults.

**Intussusception.**—An intussusception is an invagination of one portion of the bowel into an adjacent part. A constricted portion is “swallowed” by the segment below. Sometimes more than one is present. It accounts for three-fourths of the cases of acute intestinal obstruction in infancy, but is, nevertheless, of quite infrequent occurrence. Prompt recognition and suitable treatment of acute cases are essential, if life is to be saved. Chronic cases are rare.

*Etiology.*—It is most common in England and America. A family predisposition, if present, is associated with careless feeding. Males are more often affected than females, in the proportion of 2-1 under 12 years of age, and 3-1 under 12 months of age. Two-thirds of the cases occur during the first year of life, and three-fourths during the first 2 years. It is most frequent at 3-6 months of age, and in only 10 per cent. is the child over 10 years. It has occurred as early as the second day of life (E. H. Tweedy,



1906), but very few cases come on before the third month. The number then rises rapidly to a maximum in the sixth month, and falls rapidly after the seventh.

Age—Incidence of 466 cases under 1 year. Fitzwilliams (1908).

Month of Life	1	2	3	4	5	6	7	8	9	10	11	12
No. of Cases	2	2	35	63	74	84	77	35	36	25	25	8

The seasonal incidence shows a sudden rise to a maximum in March and December, i.e., at Easter and Christmas, and is well brought out by Fitzwilliams' figures :—

January ..	37	May ..	44	September	18
February ..	35	June ..	41	October ..	25
March ..	58	July ..	37	November	29
April ..	45	August ..	33	December	51

The rapidly increasing disproportion between the transverse diameters of the large and small intestine during early life is a possible predisposing cause. The colon grows in width and length. Too rapid growth is associated with perversion of function; and irregular localised contraction of the small intestine is liable to be set up by simple spasm. There is little evidence of an exciting cause. Violent cough, exertion and injury are infrequent at the common age. It is rarely due to foreign bodies, roundworm, polypus, sarcoma, tuberculous ulcer, or an invaginated Meckel's diverticulum or appendix. In quite 50 per cent. no cause can be found. Strong, healthy, well-nourished infants seem more predisposed than weakly ones, perhaps by reason of more vigorous peristalsis and more liberal diet. Diet has a definite influence. In a few instances there is gastro-enteric disorder or evidence that indigestible food has been given. This is probably the explanation of the increased frequency at Easter, Christmas and the New Year. Diarrhoea is the most common cause to which it can be assigned. Yet it does not appear less frequent in the breast-fed than in the bottle-fed, except that it is less frequent in those countries where breast-feeding is most constant. Of 46 cases under 1 year 85 per cent. were entirely breast-fed (Hirschsprung). It may occur during sleep.

*Pathology.*—For some reason or other, probably on account of spasm, a wave of peristalsis stops suddenly short at a ring-like constriction of the gut. Perhaps this is due to the lack of co-ordination so characteristic of infancy. Next, in consequence of the contraction of the longitudinal muscles, the portion of gut below the constriction passes upwards over the constricted portion. Subsequently, violent peristaltic movements, in an



effort to get rid of what has become a foreign body, force the intussusception downwards. More probably the descent is due to the paralysis of the circular fibres below and the continued contraction of the longitudinal fibres which pull up the sheath over the intussusception. The mesentery is included between the sheath and the inner layer. It is either unduly long, or becomes stretched and perhaps torn as the tumour gets larger. Being attached to the spine, the tumour forms the arc of a circle with its concavity towards the spine. The stoppage of the peristaltic wave may be due to temporary obstruction by indigestible food, or to local paralysis or atony of a portion of the gut. If the paralysis is limited, a simple descending intussusception is formed, but if it involves a considerable portion of the gut, there may be both ascending and descending intussusceptions. The apex of the invagination may remain constant, for the increase in size takes place by the outer sheath rolling over and being drawn up at the neck. Spontaneous reduction is brought about by anti-peristalsis; the circular fibres contracting beyond the apex and driving it up.

The stretching of the mesentery and pressure cause obstruction to the circulation in the vessels and strangulation leading to congestion, œdema, hæmorrhage and gangrene. The intestinal obstruction is partly due to mechanical interference and partly to the œdema. After a time the invagination becomes irreducible, at first on account of the swelling, later because of local peritonitis and adhesions between the peritoneal surfaces. These adhesions are the main cause of irreducibility in chronic cases. Occasionally the tumour as a whole becomes twisted or the prolapsed portion is nipped, increasing the difficulty of reduction.

Gangrene and sloughing of the invagination may occur in acute cases, the slough being passed per anum about the end of the second week. The slough varies in length from a few inches to several feet of intestine. Even the whole colon has been sloughed off, and the child has recovered with an ileum attached to the lower end of the rectum. Recovery by sloughing is rare in infants, and more likely to occur in older children. It is liable to be followed by annular stricture.

*Varieties.*—The common type is the *descending* variety, the upper portion of the gut being invaginated into the lower. In the *ascending* or “*retrograde*” kind the upper portion passes over the lower. Both types may be present in the same patient, and even overlap each other, though generally there is an intervening portion of unaffected gut. An *enteric* intussusception may be *jejunal* or *ileic*. It is called *ileo-colic* if the ileum passes through the ileo-cæcal valve, and *ileo-cæcal* if the invagination passes onward and involves the cæcum. These two latter types are differentiated with difficulty, and it is better to include them both under the name of *ileo-colic* or *ileo-cæcal*. The *ileo-cæcal* type is by far the most common. The percentage varies from 44 (Treves, Leichtenstein) to 89 (Wiggin). The least frequent variety is the *colic* intussusception



in the colon. *Intussusception of the appendix* may be secondary to attempts of the appendix to expel abnormal contents; of 17 recorded cases 16 were under 9 years. It is a chronic condition, and may lead to invagination of the large intestine and an acute attack.

*Agonal intussusceptions* are often found in infants under 2 years of age after death. They are generally multiple, small, descending, in the small intestine, free from any sign of congestion, and due to the act of dying.

*Symptoms* are due to local obstruction, but are not always typical. A characteristic case is that of a breast-fed infant, 3-9 months of age, well-nourished and apparently healthy, who is *suddenly* seized with severe paroxysmal abdominal pain, nausea and vomiting, the passage of blood and mucus per anum, marked prostration and a subnormal temperature.

The onset is almost invariably sudden in babies, and almost as constantly so in older children. Violent colicky pains and vomiting are the initial symptoms. Food is refused and the legs are drawn up. Occasionally diarrhoea, with blood and mucus in the stools, is the first indication. Slight fever is sometimes present. In sub-acute cases there may be merely obscure symptoms of intestinal obstruction, or less marked pain, vomiting and constipation. In chronic cases all the characteristic symptoms may be absent, and the diagnosis is based on progressive wasting, diarrhoea and an abdominal tumour.

The *pain* is severe, paroxysmal, causing agonising cries, and usually referred to the umbilicus or the site of invagination. Between the paroxysms the child may seem quite well. It is most marked during the first 2 days, perhaps only present on the first day. It is not always severe, rarely continuous, and only occasionally absent. In colic intussusception pain and collapse are marked, but the pain is less paroxysmal and more constant.

*Vomiting* is present in 80-90 per cent.; most marked at the onset; and usually not repeated until later stages of obstruction, but it may continue throughout. It is persistent, uncontrollable, immediately after feeding, and often projectile. At first the contents of the stomach are brought up, then bile, and finally, in about 20 per cent., faecal matter. Blood may be present from straining. Stercoraceous vomiting is uncommon in infants, rarely occurs before the third day, is indicative of complete obstruction rather than of strangulation, and is not necessarily a fatal omen.

The *stools* are very characteristic. At first, intestinal contents below the obstruction are passed in the shape of one or more normal stools. More often there is diarrhoea. Sometimes blood is passed at the onset, but usually not for 6-12 hours. Clubbe found blood in all but 6 out of 144 cases, and in 99 per cent. of them within 2-10 hours. The stools then consist of *blood and mucus* or pure blood, without a trace of faeces, faecal odour or gas; the constipation is complete. Often there are frequent



stools of this nature, perhaps several in an hour, following attacks of pain. They are liable to be ascribed to diarrhoea, if not properly examined. The hæmorrhage varies from a mere trace up to an ounce or more of pure blood, and occasionally is so severe as to prove fatal. Mucus is generally present. Sometimes the discharge resembles red paint. On rectal examination a polypoid tumour may be felt, and the finger is covered with blood and mucus.

A *tumour* is found in 80-90 per cent. of all cases, if the child is examined rectally and bimanually under an anæsthetic. In quite 50 per cent. of babies it cannot be felt, or only very indefinitely, in early stages. In a relaxed abdomen it may feel like an undefined thickening in the region of the cæcum and ascending colon. A small enteric tumour cannot always be felt. The characteristic lump is present within a few hours, rather movable, somewhat sausage-shaped, and perhaps a little curved. It is found most often in the right half of the abdomen, the umbilical region or left iliac fossa. It becomes harder and more prominent during paroxysms of pain, and from contraction under examination. It reaches the rectum very quickly, and can be felt per anum in about one-third of the cases. It may even protrude for an inch or two as a deep purplish or gangrenous lump, liable to be mistaken for rectal prolapse, piles, or polypus; and shows superficial ulceration and exudation. Per rectum the end of the invagination feels rather like the os uteri.

*Prostration* or *Collapse* is a marked feature, sudden in development and out of proportion to the symptoms. The child looks ill, pale, with anxious expression, sunken eyes, cold sweat on the forehead and cold extremities. The "abdominal facies" develops in a few hours. Occasionally there is a remarkable limpness without any special facial indication of illness. The pulse is small, frequent and feeble; and the temperature normal or subnormal.

The *abdomen* is relaxed at first, but the muscles become contracted and rigid during the paroxysms. A little local tenderness may be found over the site of the mischief; generally on the right side and associated with rigidity of the right rectus. Absence of tympanites in the early stages is of value. Later, the degree of tympanites and the contour of the abdomen depend upon the site of the obstruction and on peritonitis. Tenesmus is present, if the tumour reaches the sigmoid flexure or the rectum, and is due to attempts to expel what is practically a foreign body. The urine is diminished in amount and micturition often painful.

At first the child is restless. Gradually it passes into an apathetic or stuporous condition with increasing prostration, continued pain and vomiting, and towards the end a rapidly rising temperature. If the case is prolonged, the usual signs of peritonitis develop. Tonic contractures of the limbs and convulsions may occur at any period and terminate the attack, but death generally results from exhaustion.



Chronic cases may be ushered in by an attack of diarrhoea, and followed by paroxysmal abdominal pain for 24 hours or more. Sometimes the attacks of pain come on daily or at rather long intervals. Vomiting may be late and inconstant. The stools are constipated or loose, mixed with mucus, and passed with tenesmus. The abdomen is soft; the temperature subnormal; and a tumour rarely felt. Occasionally a tumour may prolapse from the rectum, for even 2 weeks. Thirst and wasting are often present. Some of these cases are due to invagination of the appendix.

*Diagnosis.*—There is little probability of erroneous diagnosis, if an examination is made under an anæsthetic in all cases of acute vomiting, with pain and the passage of blood-stained mucus. It is rare for the three cardinal signs, tumour, blood and collapse, to be all absent. In early cases, within 18 hours of the onset, the state of the child is often so good that the mischief is unsuspected. In others, the child is collapsed from the onset. An attack may be ascribed to gastritis because of the vomiting; or to ileo-colitis because of the stools, but in this there is no severe and paroxysmal pain. The *Signe de Dance*, absence of the cæcum from its normal position, is of a little value. After infancy the diagnosis is more difficult, for the bowels may act daily and appear normal, and examination per rectum may be negative, but vomiting is frequent and a tumour is found on abdominal examination. In chronic cases the diagnosis is based chiefly on the presence of a tumour. Incomplete cases are rare, and give rise to colic, blood and mucus in the stools, wasting, little or no vomiting, and an indefinite variable tumour. Usually there is little blood. They may last for weeks or years. Cases have to be diagnosed from enteritis, appendicitis, undescended testicle, thrombosis of the mesenteric artery, tumours, glands, polypus, and rectal prolapse. Henoch's purpura gives rise to abdominal pain, the passage of blood and mucus, and persistent severe vomiting. The vomit never becomes fæcal, and fæcal matter is generally found in the stools. Other evidence of purpura may be present. An intussusception may occur with purpura but be independent. Extensive rectal prolapse, cured by sloughing, may be mistaken for a cured intussusception.

The passage of blood is a most valuable sign of intussusception, but it is of the utmost importance not to think it necessary to wait for this before making a diagnosis. Profuse bleeding indicates considerable destruction, œdema, and paralysis of the gut. Gangrene and ulceration give rise to subnormal temperature, severe collapse and profound toxæmia.

*Prognosis.*—Spontaneous reduction is possible in early cases, and has even occurred after 6 days duration. Some cases of severe colic are due to temporary intussusception. Operation in these has shown that the intestine involved was thick and tensely congested, œdematous, and covered with flakes of lymph. Spontaneous recovery by sloughing is rare at all ages, and almost unknown in infancy. It is most likely to occur in the colic



variety. According to Treves, elimination of a gangrenous intussusception takes place in about 42 per cent. of irreducible cases ; and the death rate, among those in whom it does occur, is over 40 per cent. Such cases rarely recover completely and may develop stenosis. In infancy the affection is almost invariably fatal, if untreated within 24 hours or even within 12 hours. Two-thirds of the untreated die within a week, usually on the third to the sixth day. Older children die in the second week. Occasionally the duration is prolonged for 3 weeks. Death in acute cases is due to shock. In more prolonged ones there is general or local peritonitis or perforation. Chronic cases die from wasting, exhaustion or complications, but may live for years. The younger the patient, the worse is the prognosis.

The prognosis of operative treatment depends on the age of the child, the duration of the intussusception, and the duration and nature of the operation. The results are good, if reduction is effected on the first or second day. The mortality of cases treated by laparotomy on the first day is extremely small, less than 5 per cent. After 48 hours the outlook is much less hopeful, about 30-40 per cent. die. Statistics of operation are valueless, unless they are based on the age of the patient and the duration of the illness.

*Treatment.*—The proper treatment is by laparotomy. All other methods are extremely uncertain and very likely to involve a fatal waste of time. If a case is seen within a few hours of its onset, attempts to encourage spontaneous reduction may be made by gentle manipulation abdominally and rectally, in a hot bath or after the application of heat to the abdomen to encourage faintness and cause relaxation. Irrigation with warm water may be tried. In the meantime all necessary preparations for operation must be made. No food is given by the mouth, and purges are injurious. If the diagnosis has been made, a small dose of tincture of opium may be given to relieve pain, relax spasm and lessen peristalsis. It must be used with great caution, as it is liable to cause paralytic distension of the gut, and it masks symptoms.

If operation is postponed until reduction is impossible from swelling, adhesions or gangrene, it is almost invariably fatal. It should be done as early as possible in every case, no matter the age of the child. Such a line of action may lead to unnecessary operation in the rare cases which might undergo spontaneous reduction or be reduced by an injection. Even so, such a result is amply compensated by the number of lives saved, for a fatal issue is constantly due to the delay involved in carrying out other treatment and waiting to see if it has proved successful. Preliminary irrigation is justifiable to reduce the size of the tumour and render manipulation more easy. The usual precautions must be adopted to minimise shock and prevent extrusion of the bowels. The room and table must be warm ; the limbs wrapped in cotton wool and the body exposed as little as possible ; a minimum dose of anæsthetic given ; and the operation done



as quickly as possible. A small incision through the rectus muscle, in the right semilunar line, or in the median line may enable the intussusception to be reduced in situ. To save time it is generally advisable to make a larger incision and effect the reduction outside the abdomen. A certain amount of force is justifiable under the age of 2 years, because of the enormous mortality of resection. After the operation the child can be given a rectal feed, and should be fed by the mouth as soon as it recovers from the anæsthetic. Opiates must be used with great caution. Give castor oil in 48 hours if the bowels have not acted, and strychnia if necessary. There is generally some rise of temperature for a few days, and the child recovers if it survives 48 hours. Death may result from shock, the effect of the anæsthetic, toxæmia, or persistent paralysis of the gut. Recurrence is rare. Occasionally the temperature rises considerably, even to 107° F., from intestinal toxæmia, and may be fatal. A rare occurrence is the passage of blood and mucus without fæcal matter in the second week after operation, with anorexia, perhaps slight vomiting, and no fever. Possibly this condition is allied to Henoch's purpura. No recurrence is found at operation. Expectant treatment must be adopted. Rupture of the wound and prolapse of the gut is a rare sequel.

If the intussusception is irreducible, or if the bowel shows signs of gangrene, or its moribund condition is indicated by the loss of gloss, absence of bleeding when pricked and inability to reduce the congestion by massage, other methods must be adopted. In irreducible cases the best results have been obtained by resection and end-to-end anastomosis of the gut, but the operation is generally fatal from shock or exhaustion. It is hardly ever successful under 2 years of age, though recovery has taken place even at 3 months. Similar treatment is necessary for gangrene or rupture during manipulation. Other methods include incision of the sheath and removal of the invagination, short circuiting, and the formation of an artificial anus. A second operation to close the anus may be fatal.

Apart from operation recourse must be had to inflation, injection and irrigation. It is extremely improbable that any variety of intussusception, except the colic one, can be reduced by this means. The great majority are of the ileo-cæcal kind. The bulk of the intussusception may be reduced, but an inch or more remains, and its existence cannot be diagnosed until the child has been allowed to come round from the anæsthetic and the general symptoms watched for a further period. Even if reduction is complete, an engorged and œdematous gut may still suggest its presence. The treatment creates uncertainty and serious waste of time. It rarely causes complete reduction, and if the slightest invagination is left, recurrence occurs. This is by no means uncommon within 24 hours, and multiple recurrence of colic intussusceptions has been reported. If the treatment fails, as it generally does, the child has to undergo further anæsthesia. There is a small risk of rupture of the gut, for it is impossible to gauge the amount



of pressure and to exclude the presence of serious changes leading to softening of the gut. It is useless if there are adhesions, and if there is much bleeding or severe collapse. Symptoms may persist afterwards from failure or from intestinal palsy. There are few recoveries under this treatment, even in the early stages, at any rate after 12 hours duration.

For injection or inflation the child is anæsthetised, placed on the back with the pelvis well raised and the thighs flexed, and is inverted from time to time, to get the assistance of traction. A hand-bellows or Higginson's syringe attached to a catheter is used to inject air, and a funnel and tube held not more than 2 feet above the buttocks for the injection of fluids, such as salt solution, barley water, milk and water or oil, at a temperature of 100-105° F. The process must be done slowly, in 15-20 minutes, and be assisted by manipulation. Reduction is indicated by the escape of fæces and gas, disappearance of the tumour, and return of the abdomen to its normal contour. Afterwards, the child is given a small dose of opium, light diet, no purgative, kept quiet, and carefully watched for signs of recurrence.

**Hernia.**—Hernia occurs at one or other of those parts of the abdominal wall which are structurally weak or incomplete, especially the umbilicus, and the inguinal and crural canals. Some surgeons hold that in every case there is a precedent congenital peritoneal pouch. This is probably true of inguinal hernia, but it hardly explains those cases in which there is no peritoneal pouch, e.g., hernia of the cæcum and ventral hernia secondary to operation. Direct inguinal hernia is also a protrusion in the lower part of the semilunar line, through Hesselbach's triangle, and has no sac. It is very rare in children. Frequently the divaricated recti permit the linea alba to be stretched and thinned; it will then protrude as a ventral hernia. This common condition, peculiar to children, is congenital or acquired, and the hernia has no neck. The multiplicity of herniæ and the frequency of the ventral protrusion suggest a common origin in intestinal fermentation and distension, rather than a congenital sac. The chief predisposing factors in inguinal hernia are the large internal abdominal ring and the weakened muscles guarding it. The chief exciting factor, probably of more importance than the predisposing congenital factor, is the increased intra-abdominal pressure so frequently seen in rickets, consequent on abdominal distension due to the intestinal fermentation and weakened muscles.

One-third of all cases occur under 14 years of age (Coley). In children two-thirds are multiple and one-third single (Corner). It occurs in from 5-10 per cent. of all children, and is much more common in males, especially in the first year of life.

*Inguinal Hernia* is a common variety in early life and almost invariably oblique. It is often bilateral. Nine-tenths occur in males. In males it is 3 or 4 times, and in females twice as frequent on the right side as on



the left. The vaginal process closes later on the right side. It depends on total or partial patency of the peritoneal prolongation known as the processus vaginalis or funicular process. The walls of the inguinal canal are not primarily at fault. Under normal conditions of development the testis descends into the scrotum, carrying with it the funicular process. From this process the tunica vaginalis is cut off by obliteration of the narrow neck of the sac. In the common form of inguinal hernia the funicular process remains open, continuous with the peritoneal cavity, and forms a sac into which the abdominal viscera can descend. In 10-20 per cent. it communicates with the tunica vaginalis. The true congenital inguinal hernia is spoken of as total or partial funicular, according to the degree of patency of the process. "*Congenital*" means that the sac exists at birth, not that the hernia does. The sac is congenital, the hernia may be acquired. The sac may be present throughout life without any hernia forming, but the predisposition to it remains. Obliteration of this funicular process may be merely delayed and may take place subsequent to birth. The *partial congenital* or *acquired* variety is similar to the acquired hernia of adults, in that its sac is cut off and separated from the testicle and tunica vaginalis. It is a common type in infants, and is spoken of as *Incomplete*, as opposed to the *Complete* or *total* variety when the tunica vaginalis is also patent. Possibly it is always due to a congenital defect, namely, the persistence of a diverticulum, a portion of the processus vaginalis, which has not been retracted into the abdominal cavity. If truly acquired, it must be ascribed to abdominal distension and weakening of the wall, and greater importance attached to these factors than to patency of the processus.

*Congenital Hydrocele* is also due to patency of the processus, though it may be narrowed at its neck like the neck of a bottle. Through the channel fluid passes into and out of the abdominal cavity. It flows back slowly into the abdomen while in the recumbent posture, and returns when the erect posture is assumed. The swelling is translucent, dull on percussion, empties slowly without the gurgle produced on reduction of the hernia, and on its return fills up from the bottom. If it is shut off, it forms an encysted hydrocele of the cord. Infantile hernia shows a certain degree of translucency and carelessly may be mistaken for hydrocele. From its pathology it is obvious that local treatment of the hydrocele by painting, tapping, etc., is futile. Many get well without treatment, for the obliteration of the sac may be merely delayed. Others can be cured by truss; or by operation, if the condition is still present at 4-5 years of age.

*Hernia and Phimosis.*—Undue importance has been attached to phimosis as a factor in the causation and maintenance of hernia; partly because hernia is more common in boys, but this is due to the descent of the testicles. Severe phimosis is frequent without hernia and circumcision alone will not cure hernia; nor is hernia less frequent among the



circumcised than the uncircumcised. Straining on micturition is rarely due to a minute orifice in the foreskin and, unless there are extensive adhesions, ballooning of the foreskin quickly attracts attention long before hernia can be produced. If there are straining and pain, they are more often due to a narrow meatus than to phimosis. Crying and straining rather tend to strengthen the fibres of the internal oblique and transversalis muscles, approximate the anterior and posterior walls of the inguinal canal, and reduce the tendency to hernia. Such straining may encourage hernia, if there is abdominal distension and a patent processus vaginalis keeping open the inguinal canal.

*Malnutrition and Bad Feeding.*—The majority of cases are seen in badly nourished, unsuitably-fed children. It is quite common to find a hernia in severe cases of marasmus.

*The Contents of the Sac* in order of frequency are small intestine, cæcum and vermiform appendix, bladder, omentum, sigmoid flexure, and rarely a Meckel's diverticulum (Littre's Hernia). The hernia is often associated with encysted hydrocele of the cord. In females an ovary and fallopian tube may be present, sometimes in conjunction with congenital malformation. Occasionally the sac is tuberculous.

*Diagnosis.*—An inguinal hernia is higher in the abdominal wall than in adults. It is more inguinal than scrotal, and not always obvious or induced by straining. It forms a swelling in the groin, tense on straining, reducible, disappearing during sleep, and often translucent. Irreducible hernia is rare. Hernia of the cæcum on the right side is frequently incarcerated. The testicle may be undescended. An appendix in a hernial sac may simulate encysted hydrocele of the cord.

*Treatment.*—Remember the tendency to spontaneous cure. Medicinal treatment is directed to careful dieting, so as to cure abdominal distension and improve nutrition. Grey powder and rhubarb with soda are of great use. If the hernia keeps coming down, the child should be kept in bed with the legs and pelvis well raised for a week or two. The value of *truss treatment* consists in keeping the sac empty, preventing distension of the surrounding structures, and perhaps setting up adhesive inflammation about the neck of the sac and thus causing obliteration. Unless it sets up inflammation, a truss does not cure, but it prevents prolapse and dilatation of the neck of the sac, and gives better opportunity for the normal processes of obliteration. The canal becomes more oblique as the child grows older. All benefit is lost if once the hernia comes down, so the truss must be kept on day and night. The ordinary soft worsted or wool truss is of little value. A rather better one consists of a 2-in. flannel bandage, with a pad of boracic lint over the inguinal canal and external ring. These trusses can be used temporarily for slight hernia in private practice, but never for more than 6 months. The spring truss, with a pad distended with air or water and covered with indiarubber, is better. The skin must be bathed with



weak alcohol and water to prevent chafing. It is harmless in girls, but it may do injury to boys by pressure on the spermatic cord, setting up orchitis, atrophy and fibrosis. These results are most liable to happen if the hernia is of the complete congenital variety. In the incomplete or acquired variety the treatment is more satisfactory. It is, therefore, of great importance to recognise the difference between the two types. The former is often associated with congenital hydrocele or imperfectly descended testicle. Objections to the truss treatment are numerous and its management troublesome. A truss never fits for long on account of the rapid growth, and if it does not fit it is useless and dangerous. It has to be continued for several years and, even then, the sac remains and is a potential danger in later life. In mild cases truss treatment must be continued for 2 years. *Operative treatment* is far more satisfactory for boys. If it is done early in life there is no malformation of the ring, and it is sufficient to ligature the sac at the level of the internal ring and remove it. Higher up there is a risk of injuring the bladder. The early operation is done at any age previous to the onset of the first dentition, preferably after the third month and before weaning. It is a complete cure, if the sac is removed. A radical operation is not necessarily a radical cure, but recurrence is rare. The chief objection to it is that many cases are cured by truss treatment continued for 1-2 years, though a potential sac remains. For this reason many surgeons refuse to operate before the age of 4 years; but the early operation is steadily becoming more popular. The objection that it is difficult to obtain asepsis is absurd and untrue. The mortality is extremely small, and a cure is effected in 2 weeks. It is very suitable if there is difficulty in applying the truss properly or inability to afford prolonged treatment. Operation is necessary for a large hernia, which cannot be controlled by truss; for femoral, irreducible, or strangulated hernia at any age; for hernia with reducible hydrocele, if a truss is painful; for incomplete hernia with imperfectly descended testicle; and if the hernia is persistent.

*Strangulated Inguinal Hernia.*—More than 100 cases have been recorded under 1 year of age; a small number in comparison with the great frequency of hernia. Fifty per cent. of those in the first year of life are under 3 months of age; and of these one-fourth occur in the first, one-fourth in the second, and one-half in the third month. In a case of Kellock's (1902), a boy aged 10 weeks had bilateral strangulation, with an interval of 15 days between the two operations. The symptoms are more gradual in development than in adults, because of the greater elasticity of the inguinal canal and the absence of chronic thickening of the neck of the sac. There is the usual history of a hernia, perhaps treated by truss; of the rupture coming down and staying down; and the development of vomiting, constipation or the passage of bloody mucus, and frequently suppression of urine. There is a tense, painful, tender, irreducible local



swelling, perhaps resonant and with no impulse on cough. It may be so transparent as to simulate hydrocele. In later stages there are redness and œdema of the skin, general malaise, lividity and collapse. The mortality of operation is very small, if operation is done in reasonable time, for the diagnosis is usually made before gangrene has set in. Stiles had no death in 15 cases. The baby will take the breast a few hours after operation. In the treatment of irreducible hernia a gentle attempt at reduction by taxis should be made, with the child in a hot bath or suspended so that it almost stands on its head. This is by no means uncomfortable, and the steady drag of the intestine may disengage an irreducible and even strangulated hernia.

*Femoral Hernia* is much more rare. Bull and Coley (1905) had only 35 in 1,424 cases of hernia in children, 1,366 being inguinal. It may be bilateral. Stiles had only one in a series of 360 cases (1904).

*The Appendix and Hernia.*—It is not very uncommon to find the appendix and sometimes portions of the cæcum in a hernial sac, and not infrequently such a hernia becomes strangulated. The appendix may be attacked by acute inflammation while in its abnormal position.



# CHAPTER XXIX.

## THE RECTUM AND ANUS.

*Malformations — Fæcal Incontinence — Fissure — Spasm — Polypus — Prolapse — Proctitis — Hæmorrhoids — Fistula — Abscess.*

Examination of the rectum and anus often affords valuable and essential evidence of local mischief, and sometimes of affections of other organs. The rectum of the infant admits the little finger. The sigmoid flexure is loosely attached, and it may be possible to palpate the kidneys and under the surface of the liver. Examine for polypus or foreign body in cases of diarrhœa and hæmorrhage. Other affections which may be recognised by rectal examination are fæcal accumulation, intussusception, enlarged glands, an appendix abscess or inflamed appendix, the matting of tuberculous peritonitis, localised gonorrhœal peritonitis, salpingitis, ovarian cysts and tumours, and enlarged spleen. It is sometimes of value in the differential diagnosis of renal and other tumours. It is almost needless to add that the examination must be carried out with extreme gentleness, bimanually, and frequently under anæsthesia.

**Malformations.**—In the consideration of malformations of the rectum and anus it is necessary to investigate the development of the hind end of the body, including the urethra and vagina, i.e., of the rectum and urogenital passages. Three demonstrations on the subject by Keith (1908) contain the most recent observations on the subject. Keith found 37 specimens of malformation of the rectum in the museum of the Royal College of Surgeons and 77 in other London hospital museums. These may be contrasted with Curling's 100 cases (1876) in the following table, showing the frequency of the different varieties.

MALFORMATIONS OF THE RECTUM.

GROUPS.	KEITH.	CURLING.	TOTAL.
A. Males—			
1. Rectum opening in urethra .. ..	33	26	59
2. Rectum ending as cord at or above base of prostrate .. ..	7	38	59
3. Rectum ending as cord at site of proctodæum .. ..	7		
4. Rectum ending blindly at procto- dæum .. ..	7		



MALFORMATIONS OF THE RECTUM—continued.

GROUPS.	KEITH.	CURLING.	TOTAL.
B. Females—			
1. Rectum ending in vulva or vagina..	6	11	17
2. Rectum ending as cord above upper fornix of vagina .. ..	5	19	39
3. Rectum ending as cord at upper fornix of vagina .. ..	3		
4. Rectum ending as cord on vagina below upper fornix .. ..	2		
5. Rectum ending blindly or as cord at proctodæum .. ..	10		
C. Miscellaneous—			
1. Imperfect .. ..	6	6	7
2. Imperforate rectum in females with male form of ext. genitals.. ..	6		
3. Rectum opening abnormally ..	1		
4. Rare malformations.. ..	5		
5. Abnormalities in domestic animals	16		

In the process of development the hind-gut grows backward and loses its connection with the cloaca, but the other cloacal orifice, the allantoic opening, persists and forms the urogenital sinus and finally the urethra. Into this sinus there open the bladder in front and the Müllerian ducts behind.

Apparently the orifice of these ducts is lost in early foetal life and develops again later. The vagina appears as a solid rod-like structure in the tissues between the urethra and the hind-gut, now the rectum. It is formed by an active tubular downgrowth from a portion of the wall of the allantois, and the hymen is due to incomplete absorption of the terminal portion. Atresia is the result of failure of this downgrowth to develop.

The male urethra, as far as the bulb, is equivalent to the female urethra, which for a time is obliterated as a tube and is subsequently regenerated. The penile urethra is formed by the desquamation of a plug of cells invading the penis. Hence the urethra in foetal life is for a time imperforate, and if the condition persists the bladder bursts from internal pressure, urine being secreted in utero, and ectopia vesicæ results. Thus, there is a stage of imperforation in all the posterior orifices, and this stage may be persistent and give rise to various defects, more especially in the rectum.



An imperforate urethra is the least common, for foetal use is made of the channel. It causes great distension of the bladder, ectopia vesicæ, umbilical fistula and epispadias, provided the kidneys develop. Atresia vaginæ, ectopia vesicæ and imperforate anus may be combined. The hymen is almost invariably present and may be persistent.

According to this theory, that of Wood Jones, the urethral orifice of the rectum is the original anus, and the portion of the rectum between that and the perineal anus is a post-allantoic prolongation of the original rectum, or hind-gut. Keith brings forward evidence (*British Medical Journal*, 1908) which makes this hypothesis unsatisfying. Reference to his papers must be made for further details of what is a very complicated process of development. According to this writer it depends on modifications of the cloaca for sexual purposes. Mall (1908) found, from the results of the examination of 163 malformed embryos, that the chorion invariably showed evidence of uterine inflammation; and he regards this as the cause of foetal malformation.

The *anus* is formed from the proctodæum by the absorption of a definite cellular mass, the "bouchon cloacal" of Tourneux, a specialised part of the "anal membrane," forming the anterior wall of an internal cloaca at the junction of the allantoic stalk with the hind-gut. The anal canal, and both the internal and external sphincters, arise in this way, independently of the bowel, and the rectum terminates at the entrance into the anal canal. Certain malformations arise from imperfect absorption. Occasionally a membrane closes the external orifice of the anus. Sometimes a thick tail-like fold of skin extends from the median raphé of the scrotum to just behind the anal opening or the tip of the coccyx. In some cases fæces pass freely, or with pain, on either side of the fold. It can be dissected off and the anus dilated. In other instances there is no anus or evidence of one, perhaps a puckered depression lined with normal skin. Or there is a well-marked median raphé and the rectum bulges down as a blind pouch to within a variable distance of the surface. Or the anus may open into a definite cul-de-sac, and the rectum bulges down the apex of the pouch, thus leaving a definite thick separating membrane about  $\frac{3}{4}$  in. from the anus. In one child under my notice this cul-de-sac was divided by a median fold of skin. Congenital stricture of the anus may be due to a membranous septum, with a small central orifice, or to a fold of mucous membrane.

In rare instances the sigmoid and rectum are absent, the colon ending in a blind pouch in the left iliac fossa; or the first and second portions of the rectum are absent, and the sigmoid and descending colon dilated. The former malformation supports the view that there is a normal extension of growth of the hind-gut backwards, and that it is due to failure in the development of this prolongation.



The *symptoms* of imperforate anus or rectum are those of increasing intestinal obstruction. The state of the anus is easily ascertained by examination; and rectal examination will elucidate the condition of affairs in obstruction higher up. In some malformations there is partial obstruction and passage of fæces by other routes. Should the rectum communicate with the urethra, bladder or vagina, meconium may be passed in sufficient amount to enable the child to live. The bladder becomes infected and severe cystitis ensues. Fæces may be found in the urine or vagina.

*Treatment* is essentially surgical, but operation is not urgent in the incomplete cases. Frequently an incision in the median line will enable the surgeon to find the blind end of the rectum, to free it for about an inch, open it and sew its margins to those of the artificial anus. The incision may pass through the peritoneal cavity, for the peritoneum may lie between the rectum and the perineum, and between the bladder and the perineum. Hence the incision may expose the peritoneum, and it may be necessary to cut through a parietal and visceral layer in order to reach the rectum. The trocar is a particularly dangerous instrument to use in attempting to afford relief. The prognosis of operative treatment is very good as regards finding the rectum, but the child often dies, even if the rectum is opened early. If the proctodæum is present competent sphincters develop. Unrelieved cases die in less than a week from gangrene and rupture of the colon.

**Fæcal Incontinence** may occur as a symptom in any serious illness, such as meningitis, fevers and prolonged wasting diseases; in affections of the nervous system, viz., spina bifida, epilepsy, myelitis and injury of the lumbar cord; from local stretching of the sphincter ani, the result of prolapsus recti, chronic constipation, impacted fæces, the insertion of foreign bodies, sodomy and operations; or from purgative drugs. Few of these causes are operative in the first three years of life. Some cases are analogous in many respects to enuresis, showing the same nervous instability; and apparently due to persistence of the infantile condition or weakness of the sphincter, whereby it yields to a very slight stimulus. Others are of congenital origin, dependent on lack of co-ordination, or on mental defect.

Incontinence is more common in boys than girls. It may be nocturnal and diurnal, and is more frequently diurnal than nocturnal only. The stools may be constipated, but are more commonly loose, offensive and slimy. Occasionally there is lenteric diarrhœa.

Cases analogous to enuresis are easily curable, generally in a few days. Those associated with diarrhœa soon yield to bismuth. Some congenital cases have persisted beyond puberty. The treatment consists in attention to the general health, diet, exercise, fresh air, cold baths, etc. Many yield to treatment by gentian and rhubarb, strychnia or nux vomica,



belladonna and bromide, or small doses of Dover's powder, gr. 1-3, t.d.s. Ice suppositories, galvanism, and local injections of strychnia are unnecessary. Food and drink should be tepid, not cold; and no irritant foods or fruits are permissible. Relapses are not uncommon, but the general prognosis is excellent.

**Anal or Rectal Spasm.**—Anal spasm is usually due to fissure; occasionally to other local irritation of the rectal mucosa, such as simple congestion. Sometimes in highly neurotic children no cause can be found, and the nervous state must be treated. It may be associated with tenesmus. The pain is severe. The infant assumes a fully extended position, with the head thrown back and the thighs strongly adducted. The duration of the attack is variable. It is treated by hot baths, injection of hot oil and, if very severe, by the injection of a few drops of laudanum. The local cause must be cured.

**Fissure of the Anus** is produced by scratching, set up by the local irritation of threadworms, eczema, etc.; by constipation, or injury by suppositories or the nozzle of a syringe. It is most frequent in the breast-fed. The common site is the posterior wall of the rectum, near the coccyx. The minute tear readily heals but, if the cause is persistent, it is constantly torn open and a small linear ulcer results. The ulcer has thickened edges, a greyish base, and exudes a little pus and perhaps a drop of blood when the bowels act. The ano-rectal mucosa is red and swollen, covered with muco-pus in bad cases. The child should be examined in the gynæcological position, with the legs elevated. Separate the gluteal folds and press the fingers on either side of the anus in order to evert the mucous membrane. Occasionally a small bivalve speculum is needed. Examination causes painful constriction of the sphincter.

Fissure gives rise to pruritus, sickening pain on defæcation and for some time after, constipation due to anal spasm for the child fears to defæcate, screaming attacks in babies, a haggard look, irritability, sleeplessness, and even pavor and convulsions. Streaks of blood may be found on the fæces. Sometimes there is spasmodic retention of urine or reflex enuresis, and reflex priapism. The pain may be referred to adjacent parts; such as the foot and hip, causing dragging of the leg, inability to walk or stand upright, and simulation of early hip disease. The gait and attitude are protective, to prevent the rubbing together of the nates.

The constipation is treated by diet, massage, mild laxatives of senna, figs, etc. Apply oil or vaseline before defæcation, or a suppository of cocaine gr.  $\frac{1}{8}$ , ext. belladon. gr.  $\frac{1}{8}$ . Keep the parts clean with vaseline and cotton wool, and touch the base of the ulcer with the solid stick of nitrate of silver, followed by salt solution, every 2 or 3 days until cured. Thomson recommends an ointment of ichthyol and tannic acid aa dr. 1 to vaseline oz. 1. Stretching the sphincter is rarely needed. It must be done under deep narcosis.



**Rectal Polypus** is not uncommon. The tumour is attached to the rectal wall by a pedicle. It causes bleeding after defæcation, and appears when the bowels act as a smooth, dark red or dusky purple swelling, like a small cherry. Blood and slime are passed after the stool. It is usually single, myxomatous or myxo-adenomatous in structure, and is sometimes mistaken for piles, intussusception or prolapsed rectum. It is easily twisted off by a wire snare or forceps.

**Prolapsus Ani and Recti.**—The mucous membrane of the anus may be everted in the form of a dusky red, irregular ring round the orifice. It appears on the passage of a constipated stool, and is readily replaced by gentle pressure with an oily rag. No treatment is necessary beyond regulation of the bowels. An *Ano-rectal Prolapse* is one of the lowest part of the rectum, with and involving the anus. *Prolapsus Recti* is a prolapse of the whole rectal wall for a distance of 2-3 ins., projecting through the anus, but not involving the anal mucosa. It is uncommon before the age of 6 months, for the child is not sufficiently wasted and the pelvic padding of fat, which supports the bowel, is still present. From this age up to the end of the third year it is quite common, because of the frequency of marasmus, attacks of diarrhœa, and weakness of the supporting pelvic muscles during early life. It may be due to the straining set up by a fæcal concretion, constipation, threadworms, rectal polypi, and vesical calculus. Constipation is, in my opinion, an uncommon cause. Sometimes it comes down in the paroxysms of whooping cough.

The prolapse comes down when the bowels act, and is easily reduced. In bad cases it will return in the intervals between defæcation, on crying or coughing. In the worst ones it remains persistently down. Thus, it may come down daily and be returned for several weeks, and the child is not brought for treatment until it has been down persistently for a week or more. It projects from the anus as a more or less cone-shaped or cylindrical protrusion, dark red or reddish purple, corrugated with transverse folds, and a round central orifice. In advanced cases it tends to bend forward. It often becomes inflamed, covered with mucus, ulcerates superficially, and bleeds readily. If it has been down for long and cannot be replaced, constriction and necrosis of the mucosa take place at the base. It may be mistaken for polypus or intussusception.

**Treatment.**—Place the child on the side or back. Wash the tumour with cold water and reduce it by gentle pressure with the tips of the fingers, well oiled or covered with a soft oily rag. If it does not go back readily, apply ice or iced water for a short time and then try again. Should there be constipation give an enema after reduction and put the child on a suitable diet and laxatives. In bad cases it may be necessary to give chloroform or to paint the prolapse with cocaine, 5 per cent. solution, and then to dilate the sphincter by inserting the little finger through the central orifice of the tumour. After reduction insert a cocaine suppository, gr.  $\frac{1}{8}$ ,



into the rectum ; apply a pad to the anus and strap the buttocks together with adhesive plaster, or put on a T-shaped bandage.

To prevent recurrence improve the child's nutrition, cure diarrhœa, keep the bowels gently open without straining, and make the child defæcate while on the back or side, with the buttocks supported by the hands of the nurse. The squatting position, on a small seat with the legs off the ground, is the best position for older children, and prevents undue straining. After defæcation inject ice cold water, insert a cone-shaped ice suppository, or give an astringent injection of sulphate of iron grs. 2 ad. oz. 1, to reduce the venous congestion and brace up the relaxed tissues. If there is tenesmus or diarrhœa, a suppository of cocaine, belladonna or opium may be needed. Let the child lie down afterwards.

Recurrence can be prevented by keeping the child in bed with the legs suspended, as in the treatment of fractured thigh, strapping the nates together with plaster.

Should these simple measures fail recourse must be had to dusting with astringents, such as alum ; the local injection of strychnine gr.  $\frac{1}{200}$ - $\frac{1}{100}$ , twice daily, to tone up the levator ani ; Pacquelin's cautery, lunar caustic or fuming nitric acid, 3 or 4 vertical lines being drawn down the exposed mucous membrane at intervals of about an inch ; injection of 2-4 oz. of 2 per cent. saline solution into the superior pelvic rectal space, the needle being inserted midway between the anus and coccyx through the sacro-coccygeal ligament (it acts as an irritant, and causes induration of the cellular tissues) ; injections of hard paraffin, melting point 56-58° C., in the form of a ring above the anus, between the skin and mucous membrane. The bowels should be opened freely for 2 days before the operation, and a bismuth mixture given the day before. The anus must be rendered aseptic. An irreducible prolapse is so rare that resection of the mass need only be mentioned.

**Proctitis** is an inflammation limited to the rectum and not part of a general affection of the large intestine. In infants it is set up by the prolonged use of glycerine and irritating suppositories, injury or infection by enema syringes, and threadworms. Occasionally it is due to gonococcal, pneumococcal, streptococcal or diphtheritic infection. It may be a sequel of measles or scarlatina, and very rarely occur in syphilis or tuberculosis. A speculum is required for accurate diagnosis. The inflammation is catarrhal, membranous or ulcerative in type. In simple catarrh the mucosa is redder than normal, swollen, and bleeds easily. It secretes mucus freely, and discharges it in jelly-like masses or casts, unmixed with fæces. Pain and tenesmus are present on defæcation. The stools are discharged in jerks, like water from a squirt, and contain mucus and blood. Prolapse of the rectum and irritation of the skin of the buttocks and adjacent parts complicate the case.

In the membranous form the membrane may be seen on the wall of



the rectum, on examination with the speculum ; on the gut if it is prolapsed ; or in the stools.

Ulceration is usually superficial, secondary to catarrh, and heals readily. Deep follicular ulcers are sometimes found as part of a follicular colitis. Tuberculous and syphilitic ulcers are rare. Ulceration causes greater bleeding, a variable amount of pain, tenesmus if it is near the anus, and pus in the stools in chronic cases.

*Treatment.*—Attend to any local cause which may be present. Get rid of worms. Give injections of warm bland fluids, such as starch and water, saline solution, olive oil and lime water, or boric acid lotion. Later, give astringent injections, e.g. 2 per cent. solution of hamamelis or tannic acid. If there is ulceration, insert a speculum and apply nitrate of silver solution, 0·5 per cent. strength. Give cocaine or opium suppositories for pain and tenesmus ; alkalies by the mouth, if the stools are acid ; mild purgatives, a milk diet, and rest in bed.

**Hæmorrhoids** are not often seen, partly because they are not looked for. They may be found in the first few months of life, but are rare before the fourth year. External piles are due to constipation. They are generally small, may cause pain on defæcation and slight bleeding. Internal piles are sometimes present without pain or other signs. In a few cases I have seen a single purple soft pile, which would come down on the child straining, but not causing any discomfort or bleeding. Such piles disappear in time and give rise to no trouble. Treatment is rarely necessary. A mild astringent ointment can be used, and it is important to keep the bowels gently open. The *Congenital Pile* is a fibro-fatty structure ; a malformation and not a true pile. It is a small pile-like tumour, the size of a large pea, in the middle line at the anterior margin of the anus, on the perineum.

**Ischio-rectal Abscess** is not uncommon, even in infancy. It is due to lymphadenitis, secondary to proctitis or local infection. The abscess heals quickly after incision. I have known it take over a fortnight to get well. It rarely burrows, sloughs or ends in fistula.

**Fistula in Ano** is rare in infants and children, and usually tuberculous ; occasionally secondary to fish bones, infections, and ischio-rectal abscess. Ware (1906) has described a variety in infants, apparently of developmental origin, due to inclusion of skin. It is lined with stratified squamous epithelium. A probe can be passed through it into the rectum. It gives rise to a constant discharge of moisture and fæces through the small external orifice near the anus. Cure is effected by excision of the tract of the fistula.



## CHAPTER XXX.

### APPENDICITIS.

The appendix is either a vestigial remnant or a highly differentiated and specialised lymphoid structure. It is directly continuous with the cæcum in the foetus, and later is provided with a valvular fold of mucous membrane at the orifice, the valve of Gerlach. Appendicitis is by no means common in children, but has become more frequent in later years. Not more than 10 per cent. of all cases are under 10 years of age. It is more frequent in Paris than in London; and according to Selter, of Solingen, Germany, it is 7 times more common than in adults. Diagnosis is often difficult, for the early signs are apt to be unnoticed or thought unimportant. Cases may be acute, chronic, recurrent or relapsing, latent, or fulminating; circumscribed or diffuse; catarrhal, ulcerative, gangrenous, or perforative, with or without abscess formation.

*Etiology.*—It is more common in boys than in girls in the proportion of about 2-1, and for this there is no satisfactory explanation. Neuberg (1907) found the sexes equal in 82 cases. It is rare during infancy. Death has been reported (Porak and Durante, 1902) at 21 days of age from acute peritonitis and sloughing appendix, due to a suppurating cord and urachus. Kermisson and Guimbélot (1906) collected 9 cases under 1 year, all fatal. One such case has come under my care. The causes of the recent increase in frequency have been stated to be influenza, the habitual use of purgatives, enamelled pans, iron fragments in roller flour, and chilled meat. Preserved foods and chilled meats are particularly liable to bacterial contamination. Any cause of catarrhal inflammation or congestion of the cæcum and the appendix, e.g. enteritis, is liable to set up an attack. The appendix becomes swollen, perhaps as large as the little finger, filled with mucus or muco-pus, and shut off from the cæcum. A mild attack subsides, or perhaps leads to the formation of a concretion; or the concretion may be the primary cause of the catarrh. In a few cases foreign bodies have been found present, e.g., small seeds, pins and threadworms. Santorini, in 1724, noted that the appendix was often the resort of intestinal worms.

Usually the nucleus of the concretion is a small mass of degenerated cells, bacteria and inspissated mucus. Probably most of the cases ascribed to cold, rheumatism and injury are those in which a concretion already exists and fresh catarrh has been set up. The recovery of such cases under



treatment by salicylates does not prove that they are rheumatic. The threadworms are the most common variety of worm found. Whether primary or secondary, they are liable to prolong or increase local mischief. Tuberculous and typhoid ulceration may occasionally spread to the appendix. Infection may take place from the female genitals. Malignant disease is exceptional. Injury has caused perforation.

*Pathology.*—The bacillus coli is constantly present; occasionally associated with the staphylococcus aureus, and less often with other organisms. The normal appendix is never sterile, and microbial infection is the main cause of inflammation. In the catarrhal state the inflammation may spread to the peritoneal surface and set up local or general peritonitis, apart from actual perforation. Inflammation of a pseudo-membranous type may lead to necrosis of the mucous membrane and gangrene. The catarrhal process often terminates in resolution. This may be complete; or may leave behind it adhesions, scarred tissue, strictures, or sclerosis and obliteration of the lumen. The inflammation may become chronic, in consequence of stricture and retention of secretions, forming a cyst, mucocele, empyema or concretion. Or it may end in abscess formation, the abscess being shut off from the general peritoneal cavity or setting up septic peritonitis. An abscess may form, with or without perforation of the appendix. It may be localised in the peritoneal sac, or outside the peritoneal sac and spread up behind the colon. Suppuration may extend upwards to form a subphrenic abscess, or down into the pelvis. In a girl, aged 4 years, an abscess formed below and to the left of the umbilicus. Perforation is secondary to concretion, abscess, ulceration or gangrene. It causes local abscess formation or general peritonitis. Gangrene is due to the virulence of the inflammation, or obstruction to the blood supply by twisting or kinking of the inflamed appendix. The most striking features of the disease in children are that obliteration does not occur, and that a diffuse inflammation is more common than a local one. The local plastic type of peritonitis, in which adhesions are formed and the abscess cut off from the general peritoneal character, is uncommon. When an abscess is formed it is generally, but not invariably, situated in the right iliac fossa; for the appendix may be situated in the pelvis, in the depression between the psoas muscle and the spine, in the region of the kidney, under the liver, near the umbilicus, and even in the left iliac fossa or a hernial sac. It may discharge through the abdominal wall, below Poupart's ligament, into the rectum, bowel, bladder, vagina or into the peritoneal cavity. Possibly pus can be located in the appendix for a time and discharge into the cæcum.

*Symptoms.*—Appendicitis in nurslings is of 2 types. It may be insidious in onset with foetid diarrhoea or constipation, vomiting, slight fever and wasting. This gradually passes on into a stage of increased vomiting, higher temperature, increased pulse rate, distension and peritonitis. In



the second type the onset is sudden, with pain and vomiting, high temperature, rapid pulse, severe constipation, a swollen tender belly, and general peritonitis in a few days. Cases under 18 months of age are almost invariably fatal.

The classical signs are vomiting, abdominal pain, fever, increased pulse-rate, local tenderness, rigidity and constipation. In a mild catarrhal attack symptoms may be entirely absent, or ascribed to another cause. There may be merely a little fever, nausea, slight colicky and paroxysmal pain, dyspepsia and constipation. In more severe attacks there may be found local tenderness at McBurney's spot or Clado's point, rigidity of the right rectus, absence of the abdominal reflex on the right side, flatulence, nausea, vomiting, tenesmus and diarrhoea. McBurney's spot is at the junction of a line drawn from the umbilicus to the anterior superior spine with the outer border of the rectus. Clado's point is at the junction of a line joining the two anterior superior spines, and the outer border of the rectus.

*Pain* is generally the first symptom. It varies greatly in severity. It may be severe and localised in the right iliac region. Sometimes it is referred to the right hypochondrium or diaphragm, but more usually to the umbilicus or epigastrium, and later to the right iliac region and the whole of the abdomen. There may be merely a sense of pressure in the region of the appendix. Tenderness is always present, and varies with the severity of the disease; it is not always localised at the same spot. Sherren states that it is over the skin supplied by the eleventh dorsal nerve, unless the appendix is perforated. Rigidity is limited to the lower quadrant of the abdomen in simple cases.

*Vomiting* usually subsides in a few hours, except in fulminating cases. *Diarrhoea* is more frequent in children than constipation. *Bladder* troubles are common; micturition is often painful and frequent; retention, pain over the bladder at the end of micturition and vesical tenesmus also occur, especially if the appendix is in the pelvis.

The *temperature* is raised, except in appendicular colic and bad fulminating cases. A high temperature indicates gravity, but a low one is no sign of safety. The amount of fever often varies inversely as the shock and severity of infection. The temperature may rise because the shock of perforation has passed off.

The *blood* is of no diagnostic importance in catarrhal cases. When suppuration occurs there is a reduction in hæmoglobin, of about 30 per cent., and a leucocytosis of polymorphs. Leucocytosis is also found in gangrene, perforation and peritonitis. In perforation there may be a sudden rise in the number of leucocytes. If the patient is very weak, there may be a fall. Leucopenia is present in grave sepsis, and is a bad sign if the symptoms are severe.



The *local signs* are swelling, pain and rigidity in the right iliac region, and a palpable tumour or diffused sense of resistance. Sometimes the appendix can be felt as a tender, cord-like swelling. A bimanual examination must be made. Rectal examination affords indications of pelvic peritonitis or suppuration, and of local tenderness when the appendix is in the pelvis. The tender, swollen appendix can occasionally be distinctly felt.

If there is local peritonitis, the pain and tenderness in the right iliac fossa are more severe and accompanied by greater rigidity and a more diffuse swelling, due to inflammatory matting of the parts. If it remains plastic, the temperature ranges between 100° and 103° F. for about a week, and then gradually falls after a somewhat irregular course. The swelling becomes more and more defined, forms a definite localised tumour, and disappears slowly. The other symptoms are general malaise, anorexia, diarrhoea or constipation, and perhaps vomiting.

Unfortunately mild cases of this type are uncommon, and the more serious ones end in suppuration and abscess. Pus is usually found at the end of 4 days of acute symptoms, or if the temperature still remains up. There may be distinct improvement after a few days, localisation of suppuration and slow development of the abscess. The temperature may fall, and then rise and become irregular. In many cases the temperature falls, pain lessens, the abdominal swelling becomes more defined because the abscess has burst into the bowel, and usually a loose offensive stool is passed. The local signs of abscess are more diffused pain, tenderness, rigidity and swelling; redness and œdema of the skin and fluctuation, if the abscess bulges anteriorly. Occasionally it develops slowly with few symptoms. The thigh is often flexed to relieve tension.

Many cases end in septic peritonitis. In most of these, previous mild attacks of catarrh have occurred and one or more concretions formed. Gradually the concretion ulcerates through and a small pocket of pus is shut off at the tip of the appendix, or it may rupture into the general peritoneal cavity. Frequently from some slight cause, such as strain or injury, the ulcerated patch already verging on perforation has given way and set up acute perforative peritonitis. Unless this is quickly limited by adhesions, or if the escaping matter is virulent, infective peritonitis rapidly ensues. Thus, a boy, aged 7, played football in the afternoon, went home on the top of a tramcar, and eat a large tea of buns and bloater paste. No less than three exciting causes, strain, cold and unsuitable food, were present. In the evening he was seized with abdominal pain and vomiting. He was treated for digestive disturbance, but remained unwell, constipated and feverish. On the fourth day a lump in the right iliac fossa was ascribed to fæcal accumulation. The bowels acted well after an enema; a dose of castor oil produced three good actions next day, and the lump disappeared. On the evening of the seventh day he became collapsed,



vomited and had abdominal pain. When seen next morning, he was collapsed and moribund, the abdomen was a little tense, and there was general tenderness; death took place an hour later. In a similar case, on account of the absence of localising signs and very slight indications of peritonitis, operation had been postponed. It was decided, however, to explore, and a typical condition of acute septic peritonitis, secondary to perforation by concretion, was found.

*Perforative Peritonitis* is characterised by sudden severe pain in the right iliac fossa, acute, lancinating and continuous; vomiting of the contents of the stomach and then bile; sometimes chilliness or rigor; prostration and general appearance of serious illness. The vomiting may be persistent, and become greenish and fæculent. Occasionally the perforation comes on insidiously. Local signs may be absent. The signs of peritonitis develop usually in 3 or 4 days. The acute symptoms due to perforation may have subsided and apparent improvement set in. The abdominal signs may be slight or absent, or those found in acute peritonitis. Perforation can take place suddenly and quite independently of apparent illness.

*Fulminating Peritonitis* is due to acute gangrene from virulent infection and possibly thrombosis. The symptoms are intense—more severe pain, general tenderness, early tympanites, sometimes an initial rigor. The pulse is small and frequent, temperature subnormal, and the facies of the abdominal type. Pain may cease early in the worst cases. Often there are no local signs, neither tenderness nor swelling. I have seen all these symptoms in one case of acute tuberculous peritonitis.

*Diagnosis.*—In infants under 2 years diagnosis is very difficult, because of the inability to answer questions and locate pain, and resistance to palpation. If constipation is a prominent sign, it may suggest intestinal obstruction or simulate intussusception, but the temperature is usually high and there is no bloody rectal discharge. Mild attacks at all ages may be diagnosed as appendicular colic. Such cases are due to slight catarrh, or irritation of the peritoneum by a fish bone or a pin sticking through the appendix. Attacks are liable to be confounded with gastro-enteric disturbance and colic from any cause; all kinds of intestinal obstruction; acute indigestion with fever; local causes of suppuration in the right iliac fossa, e.g., psoas abscess; tuberculous peritonitis; perforative peritonitis from other causes; fæcal accumulation, and typhoid fever (no leucocytosis). Stomach-ache is common in children who eat many odd articles of diet. It is usually acute and soon passes off, perhaps with vomiting or diarrhoea and no rise of temperature. The pain of rheumatism in one hip, or hip disease and flexion of the thigh may suggest appendicitis; so, too, those cases of pleurisy and pneumonia in which the pain is referred to the iliac region, but in these breathing is rapid and the face flushed or livid.



The main symptoms on which stress must be laid are the history of previous attacks and the onset, with vomiting, abdominal pain, fitful crying, disturbed sleep, local tenderness and rigidity, anxious face, rising pulse-rate and temperature, and discomfort on extending the lower limbs. Pain is more often absent in children than in adults. Decrease of pain can be deceptive; it may indicate improvement or toxæmia. Rectal examination can prove negative, and yet three days later a large abscess may be found. It must never be omitted, except at the onset of a mild attack. Peritonitis due to perforation is distinguished from other varieties by the history and severity. It is most likely to be mistaken for the pneumococcal or gonococcal form, both more common in girls than in boys.

*Prognosis.*—Mild cases frequently terminate in resolution within a few days. How often this occurs it is impossible to tell, for the bulk of mild cases must be overlooked, if we believe that concretion is proof of a former attack. Of the cases that are recognised and come under proper treatment, a certain number end in resolution, but the majority terminate in abscess, concretion, and local or general peritonitis. The prognosis is very bad in infancy. Recovery under 18 months of age is rare. The disease is more dangerous in infants than children, because of the greater difficulty in diagnosis and the greater liability to generalised infection in early life. Attacks are often put down to colic. Suppuration readily occurs. In even the mildest cases the prognosis must be guarded, for the clinical condition constantly does not correspond with the severity of the lesion, and acute peritonitis may supervene at any time. The possibility of recovery with a residual concretion necessitates a warning as to subsequent attacks. Yet recurrence is not common. A mild attack lasts for 1-3 weeks, and a bad one may be prolonged for as many months. Improvement is indicated by cessation of vomiting and diminution of constipation if present. A rise in temperature, pulse rate and number of leucocytes is a sign of a relapse or extension. Neither pulse nor temperature can be relied on. Even in very severe cases there may be little change in either. Coffee-ground vomiting is ominous of paralytic ileus. The most acute cases may die in 36-48 hours; usually in about a week. Those in which an abscess forms do very well under operative treatment and badly without. A favourable result may follow rupture through the abdominal wall, rectum, or vagina. More often, if left alone, death results from some complication. Quite 80 per cent. die from peritonitis, and the remainder from asthenia, pyæmia, or lardaceous disease secondary to a permanent fistula. In the fulminating cases death is due to toxæmia. It has also followed operation, from acute acidosis. Temporary improvement may result from perforation and relief of tension, or the cessation of toxic absorption, due to thrombosis or gangrene.

*Treatment.*—The child must be kept in bed, in a recumbent position with the knees over a pillow, given a liquid diet, and carefully watched.



In very acute cases starvation and saline enemata, to relieve thirst, are useful. An ice-bag, hot fomentation, or belladonna and glycerine is applied locally. Children usually prefer heat to cold. Opiates, bleeding, leeches and blisters must be avoided; so, too, purgatives, because of the consequent peristalsis; an enema of ordinary soap and water must be given. In the earliest stage of a catarrhal attack a dose of calomel, followed by a saline aperient and an enema, is very beneficial. Unfortunately it is often impossible to distinguish a mild attack from the beginning of perforation, in which treatment by purgatives is dangerous. It is much safer to avoid purgatives and to rely on enemata. Expectant treatment is only safe in mild, acute cases kept under skilled observation. As soon as the diagnosis and the type of the disease are established, small doses of opium may be given to relieve pain and to keep the intestines at rest. It is liable to paralyse the intestines and cause tympanites, and is better avoided. If used early it is apt to obscure symptoms and lead to apparent improvement, although the patient is getting worse. Hence, operation may be fatally postponed and an unduly favourable prognosis given.

The question of operation depends upon the circumstances of each case. If all patients are operated on, many cases which would recover by resolution are submitted to an unnecessary and expensive ordeal. Operation is quite as dangerous in a mild attack as in a severe one, and many recover in a few days without. On the other hand, delay leaves the patient to chance; and there is little prospect of localisation in childhood.

In addition there is a liability to erroneous diagnosis in the first 48 hours. Some lives are sacrificed to too early and others to too late interference. If signs of peritoneal irritation are present, operation is advisable. In fulminating cases there is a fair hope of recovery if operation is done within 12 hours, in a period of quiet sometimes present after the shock has passed off. In all but the mildest cases it is advisable to operate within 48 hours. After that time be guided by circumstances. In a case of moderate severity, if the pulse rate is increasing, the temperature rising, and the local and general symptoms getting worse, operate; for there is almost certainly suppuration, and possibly perforation. It is still more imperative if the temperature is falling and the pulse-rate rising. The facial expression often indicates continued mischief, and actual gangrene may have begun. If on the other hand the temperature is falling, the pulse-rate less frequent, and the local and constitutional symptoms less severe, operation may be postponed, provided a careful watch is kept on the case. Immediate operation is necessary in cases of perforation and of abscess. Increasing leucocytosis is of some value as a sign. In recurrent cases remove the appendix during a period of quiescence. Advise operation if the symptoms do not entirely disappear after an acute attack. When operating, seek for fæcal fistulæ, for in many quiescent cases the abscess has



discharged into the small intestine, and one or more fistulæ may be left. If these are not properly closed, fatal peritonitis will follow operation. The presence of a distinct lump after an attack, although the child seems well, necessitates operation. In a girl, aged 4 years, a lump of this nature was found to be a concretion on the verge of perforation. At and after operation the patient should be kept in the Fowler position, the head and shoulders raised so that the pus gravitates downward, instead of upward into the dangerous subphrenic area. In opening an abscess, it is advisable merely to incise and drain, leaving the appendix, for it is shut off by adhesions. Flushing the abdomen is apt to be injurious in septic peritonitis ; it takes time and spreads infection. Give saline injections freely in all virulent and toxæmic cases, to counteract shock, raise blood pressure, and eliminate toxins.



## CHAPTER XXXI.

### THE PERITONEUM.

*Ascites—Acute Peritonitis—Chronic and Tuberculous Peritonitis—Mesenteric Cysts—Pancreatic Cysts—New Growths.*

**Ascites.**—The area of peritoneum is about equal to that of the skin and affords a large surface through which absorption and effusion are constantly going on. An excess of fluid in the peritoneal cavity is due to inflammation or transudation. Ascites is caused by transudation by virtue of increased pressure, altered conditions of the blood and lymph, and nutritional changes in the epithelium. The last factor is most important, for probably no excessive effusion can take place without impaired nutrition. The amount of osmosis varies with the specific gravity of the fluids. Absorption takes place through the lymphatics and blood vessels; probably to a great extent by means of those lymphatics of the diaphragm which run to the anterior mediastinal glands. Simple ascites is due to backward pressure from obstructive heart or pulmonary disease, pressure on the vena cava inferior, mediastino-pericarditis, hepatic cirrhosis, thrombosis of the portal vein, or occlusion of the vein by pressure of glands or adhesions; blood states leading to malnutrition, such as leukæmic affections and renal disease; and tumours.

Hæmorrhagic ascites is rare and usually due to malignant tumours, tuberculous disease or septic thrombosis. In chylous ascites the fluid contains fat. It may be due to injury of the thoracic duct, occasionally to tuberculous peritonitis. It is very rare and of bad prognosis.

**Acute Peritonitis** is quite an uncommon affection after the first few weeks of life. In the fœtus it is due to syphilis or is of unknown causation, rarely appendicitis or placental infection, and leads to death in utero or intestinal malformation. In the newborn it is septic and results from pyogenic infection, usually through the navel. In infants it is often part of a polyserositis of streptococcal or pneumococcal origin, or secondary to ileo-colitis, vulvo-vaginitis, etc. At any time after birth the main causes are microbial infection, and perforation of some viscus or direct extension. Exposure to cold and wet predispose. Rheumatic peritonitis is of doubtful existence; such cases are probably pneumococcal. Traumatism, accidental or surgical, accounts for a few cases. The chief secondary causes are appendicitis and typhoid ulceration, with or without perforation;



perforation of gastric, duodenal or intestinal ulcers; various forms of intestinal obstruction; abscess in a lymph gland, the liver, kidney, pleural cavity or in Pott's disease. The gonorrhœal form is secondary, and the pneumococcal form generally so. Common organisms are streptococci, pneumococci and the bacillus coli; less often, staphylococcus aureus and albus, gonococcus, micrococcus lanceolatus, B. proteus, B. pyocyaneus, B. typhosus and others. Many cases are mixed infections.

*Morbid Anatomy.*—At first there is loss of lustre, and then the peritoneum becomes red and injected, serum and lymph are effused, and adhesions form. Sometimes the great omentum is the part first or mainly affected. In the plastic or fibrinous form there is little effusion of fluid. In the serous type there is little lymph and much fluid, which may be clear, turbid or blood-stained. To a certain extent the exudation of lymph is protective, for it limits the absorption of toxic products by the peritoneum. In the sero-purulent or purulent variety there are much pus and lymph, especially if it is due to perforation. The pus may become localised in the iliac fossa, pelvis, centre of the abdomen or under the diaphragm. Such an abscess may burst externally, frequently at the umbilicus; may burrow its way into a viscus, such as the liver; or may open into the bladder, rectum, vagina, or even the pelvis of the kidney. In streptococcal peritonitis the fluid is thin and yellowish, and does not tend to localisation. The local affection is a part of a general streptococcal pyæmia.

*Symptoms.*—In infants the cause of illness may be overlooked, for the symptoms are often obscure. In the newborn there may be no vomiting and little or no fever, merely some refusal of food for fear of sickness. Usually an attack commences suddenly with vomiting and high fever, round about 104° F. The abdominal pain is indicated by fretfulness, restlessness, sudden waking, drawing the hand across the abdomen, screaming, and perhaps flexion of the knees. The pain is paroxysmal or continuous, with exacerbations. Older children complain of pain when it is present. It is by no means uncommon for most virulent attacks to be quite unassociated with pain or with only a little abdominal discomfort. The amount of tenderness is very variable and that of muscular rigidity, whether local or widespread, difficult to estimate. At first the abdomen is retracted, empty, and more or less rigid and immobile. Later it becomes distended, uniformly so as a rule, and very tympanitic. There is not often much fluid, merely sufficient to cause dulness in the flanks. Occasionally a friction rub can be heard. Rigidity and tenderness vary much, and may be absent in late stages. The child assumes the dorsal decubitus with the knees drawn up. Diarrhœa is generally present in streptococcal and pneumococcal infections, and is always more common than in adults. Retention or incontinence of urine and dysuria are frequent.

Vomiting usually stops, but may continue throughout and become fæculent towards the end. The features are pinched, haggard and anxious.



The characteristic *Abdominal Facies* is sometimes absent even a few hours before death. Respiration is thoracic, shallow, and increases in frequency with the distension. The pulse rate is more important than the temperature; the pulse is frequent, small, soft and running. Leucocytosis rapidly develops in perforation cases. In these the temperature is low at first, and rises after the period of reaction. Prostration becomes more and more profound, the extremities cold, and hiccough, collapse and exhaustion end the scene.

*Pneumococcal Peritonitis* is more frequent in children than in adults, and in girls than in boys, in the proportion of 3-1. Annand and Bowen (1906) collected 91 cases, including 16 of their own, in children under 15 years of age. They regarded 47 as primary and 44 secondary. Several cases have been recorded since. In the primary cases infection is probably carried by the blood stream. Of the secondary cases three-fourths are due to lung mischief, and the remainder to otitis media, sore throat, umbilical infection, and possibly a pneumococcal appendicitis or enteritis, or infection viâ the genitals.

The peritoneal symptoms may be relatively slight or severe. The exudation is profuse, rich in fibrin, very coagulable, greenish yellow, and not offensive. Fibrinous layers of lymph and jelly-like masses of pus are often present. Adhesions and early localisation, generally in the lower part of the abdomen, are common, but the inflammation may be diffuse. Rarely the exudation is hæmorrhagic. It may be offensive from post mortem decomposition. The lung affection, if present, may occur before or after, or be coincident with the peritonitis.

*The symptoms* are those of an acute peritonitis, usually less severe and more often associated with diarrhœa. Its onset is generally sudden, with vomiting and diarrhœa, colicky pains, especially in the lower part of the abdomen, and high fever. In a few days the symptoms become less severe, but diarrhœa persists, meteorism develops, and pus collects. Pain may be great and generalised, and the temperature hectic. The abdominal distension is sometimes a prominent feature. Two varieties can be distinguished. There is a diffuse, violent, persistent type with prostration, cyanosis, rapid pulse, abdominal facies, and rapidly fatal end. The other is acute in onset, but becomes chronic or circumscribed, and the pus is encapsuled, forming an *Abdominal Empyema*. The abscess is usually sub-umbilical, median or lateral, and perforates the umbilicus in about 4-6 weeks, if untreated. It may discharge through the vagina or bladder. It may fill the whole abdomen. These cases in their general appearance simulate chronic tuberculous peritonitis, and the local abscess had generally been regarded in the past as a tuberculous abscess.

*Gonococcal Peritonitis* has been recorded as early as 5 months of age. It is due to infection spreading along the vagina, uterus and tubes. It is often localised, causing salpingitis, which may be felt per rectum. The



gonorrhœa may be acute or chronic, and is seldom of a severe type. The onset is usually acute with frequent vomiting, frequent micturition, alternate diarrhœa and constipation, and a temperature up to  $104^{\circ}$  F. The pulse is frequent, small and weak; the extremities cold and cyanosed; and the abdomen tender and tympanitic. The child lies with the legs crossed or drawn up, screams with pain, is somewhat delirious, and is soon very gravely ill. An attack lasts from 1-8 days, and the acute symptoms may subside rapidly for the vitality of the gonococcus is small in cellular tissues. A gonococcal peritonitis is usually localised, and a rapidly fatal generalised variety is due to a mixed infection. The diagnosis is difficult because it is unsuspected. A simultaneous occurrence of peritonitis in sisters is very suggestive. It is liable to be mistaken for appendicitis. The prognosis is good and relapse is exceptional. Laparotomy is said to seriously aggravate the condition.

*Diagnosis.*—Peritonitis at the onset must be distinguished from colic. Later on it may simulate cholera, dysentery, and acute colitis or entero-colitis. Pneumonia and pleurisy have been erroneously diagnosed as peritonitis or appendicitis, and the abdomen opened. Such a mistake ought not to arise, for the breathing is rapid and out of proportion to the pulse-rate and temperature, the temperature is generally higher, cough sometimes present, and the rigidity of the abdominal muscles usually relaxes during inspiration and on steady pressure. Henoch's purpura, acute intestinal obstruction, appendicitis, and perforation of an abdominal viscus may be mistaken for peritonitis, of which they are often the cause. Differential diagnosis of the different types is fairly easy. In the newborn the disease is almost always streptococcal. The gonococcal variety occurs in girls, usually over 4 years of age, vulvo-vaginitis is almost invariably present, and the pain at first is often localised in the lower part of the abdomen. The presence of pulmonary disease, herpes labialis, and green loose inoffensive stools indicate the pneumococcal type. A primary pneumococcal affection is not improbable in all those cases of acute onset, without a history indicative of appendicitis or a perforative lesion. I have known tuberculous peritonitis so acute in its onset as to suggest perforation and secondary peritonitis. A boy, 12 years old, was seized with vomiting and abdominal pain, and on the third day was almost moribund from collapse and toxæmia. On laparotomy the intestines were found covered with tubercles and much fluid was let out, and the patient recovered. Some of the acute cases of the pneumococcal type may be mistaken for typhoid fever, if a leucocyte count is not made. Rectal examination is often of assistance.

The *Prognosis* is very bad because of the large area for the absorption of toxins. In localised cases the inflammation is protective. The pyogenic form in infants ends fatally in 2 or 3 days, but older children live for about a week. After that the outlook is more hopeful, for the mischief may



become localised. The diffuse pneumococcal type is generally fatal, and the circumscribed ends in recovery. Sero-fibrinous effusions are the least dangerous, and purulent ones the most fatal, for they are often due to perforation, especially of an appendix, and terminate in 6 or 7 days. The temporary improvement after perforation must not be regarded favourably, as it is only the result of reaction, and the onset of peritonitis is indicated by increased frequency of the pulse and abdominal distension, and progressive illness.

*The Treatment* varies with the nature of the case. In acute general peritonitis, not due to perforation, cold or heat may be applied locally, e.g., spongiopiline wrung out in hot or cold water, an ice-bag, bran poultice, or turpentine stupes. Give a saline purge, if there is constipation and no appendicitis. Use the common remedies for vomiting (p. 252). Give opium gr.  $\frac{1}{20}$  -  $\frac{1}{10}$ , every 2 or 3 hours, to control the pain and peristalsis. It may keep up the vomiting and its effects in minimising symptoms must be remembered. Feed the child carefully with peptonised milk, whey, white of egg, and other easily digestible foods. In severe vomiting it may be necessary to omit food for a time. Give a liberal supply of fluid by various channels, and brandy and champagne as stimulants. Laparotomy is necessary in pneumococcal cases when pus has formed, in those due to perforation, and in most septic ones. The removal of fluid leads to the effusion of fresh serum with greater germicidal power than in that removed. If on laparotomy there is a general diffused peritonitis and much purulent fluid, seek and remove the cause if possible and drain. Do not mop, wash or irrigate. Keep the patient in a sitting posture and give saline injections. On the whole laparotomy must be recommended with caution, except in the pneumococcal cases and those due to perforation. An abscess must be treated as a chronic abscess, opened and drained.

**Chronic Peritonitis** is generally tuberculous. A few cases are secondary to acute attacks, visceral disease, vaginal infection, cold or injury. Possibly some are syphilitic. In one case, prolonged and fatal, it was a sequel of acute ptomaine poisoning, but no organisms were found in the peritoneal exudation.

*Sub-diaphragmatic or Sub-phrenic Abscess* is an accumulation of pus under the diaphragm and above the liver, or, rarely, the spleen. It is due to pneumonia, disease of the liver, extension from tuberculous cavities, and occasionally appendix abscess. It is usually mistaken for empyema, or pneumothorax if it contains air. The physical signs are those of empyema, and it is treated by incision and drainage.

**Tuberculous Peritonitis.**—In acute miliary tuberculosis the peritoneum may be studded with grey tubercles, without the spread of inflammatory changes to the peritoneum, and death occurs before the onset of any definite abdominal symptoms. If the illness is more prolonged the tubercles



undergo caseous degeneration and set up peritonitis and exudation. In rare instances acute peritonitis is due to this cause, and the tuberculous affection limited to the abdominal cavity. More commonly we have to deal with a chronic form divisible into the following groups, viz. :—

- (1) Adhesive, fibrous or plastic peritonitis.
- (2) Peritonitis with effusion, of serum or pus.
- (3) Caseous or ulcerative peritonitis.

The source of infection is doubtful. It may be conveyed by lymphatics or blood vessels from distant parts; it may spread from a broken down gland or from the Fallopian tubes; or be due to the breaking down of a tubercle in the wall of a small artery, and dissemination to the parts supplied by that artery; or secondary to tuberculous ulceration of the intestines. The etiology is that of tuberculous disease generally. Malnutrition, unsuitable food, and insufficient clothing of the abdomen render the peritoneum susceptible to infection.

It occurs at all ages, but is most common at 2-4 years. Almost half of 306 cases under 15 years of age were between 3 and 7, and the sexes were about equal (Faludi, 1905). In later life it is very much more frequent in females or, perhaps, is more readily recognised in them because they are more liable to be operated on for abdominal conditions.

*The symptoms* vary somewhat with the type of disease, and include anorexia, wasting, variable degree of fever, abdominal distension and ascites. The superficial veins are dilated. The distension depends upon tympanites and fluid, so the degree of dulness varies. Fever runs no definite course; it may be hectic in the caseous type. Anæmia, sweating and prostration are often present. In the ascitic type tubercles are present in all stages, and there are few adhesions. A localised serous effusion may be mistaken for a cyst. Any variety may be associated with scattered lumps, due to tuberculous mesenteric glands. In the caseous form caseous masses are felt. Sometimes the omentum stretches across the abdomen as a rolled up, hard, irregular, nodular, caseous band. In the adhesive variety there is little or no fluid, and a more or less general matting together of the intestines. It is the commonest type in children, and may be secondary to the more acute form with effusion of serum. The fluid is usually clear, occasionally sero-purulent or bloody, less commonly purulent. Effusions of pus are generally due to rupture of a caseous gland or caseous mass in the mesentery, or due to secondary infection. It may be localised as a chronic abscess.

In the caseous variety the intestines are matted together by adhesions, with masses of caseous material covering and separating the coils. In the milder degrees there may be only one or more semi-solid, indefinite lumps or a general doughy feeling of the abdomen, with patches of irregular dulness and resonance. This form is differentiated with



difficulty from the adhesive type with local accumulation of fæces and localised flatulent distension.

The *course* of the disease is slow and irregular. The adhesive type is tedious and may last for 12 months, and there may be some effusion of fluid. In peritonitis with effusion of serum the acute stage lasts from 4-8 weeks, and sometimes longer, the fluid then being re-absorbed, and the case assuming the characters of the adhesive variety. In the caseous type the course is somewhat similar, if there are only 1 or 2 localised masses, but in the diffused caseous form the patient steadily gets worse and dies. Consequently, the prognosis depends on many different factors, such as the age of the patient, the hygienic and social surroundings, the pathological state, the physical condition, and the presence of the various complications and sequelæ that may arise. The adhesive type is rarely fatal, unless it produces intestinal obstructions by adhesions. Constipation is often troublesome. Some cases die from meningitis, tuberculosis of the lung, or miliary tuberculosis. The prognosis is worst in the caseous variety. The most fatal complications are intestinal ulceration and general dissemination. As long as the disease is confined to the peritoneum, the prognosis is good, except in the caseous variety. The gloomy views of past physicians and surgeons are not supported by recent experience. Probably quite 50 per cent. of hospital cases recover, and, I believe, in private practice the percentage of recovery is considerable higher, if they are properly treated. The prognosis is worst in the youngest cases.

The signs of the patient getting worse are continuous fever, rapid wasting, diarrhœa and vomiting. The prognosis is unfavourable in the presence of lung infection, recurrent exacerbations, pleurisy, albuminuria, dropsy, localised suppurations, caseous mesenteric glands, or intestinal obstruction. A rare complication is pigmentation due to pressure of glands on the adrenals.

*Diagnosis.*—Undoubtedly many mild cases are undiagnosed. It is impossible to be certain that some of these are not due to simple flatulent distension from other causes. Such distension in apparently healthy children, especially when accompanied by vague abdominal pains or other symptoms referable to the intestinal tract, must be regarded as possibly tuberculous. Cases are sometimes diagnosed as ovarian cysts or tumours, hydatids, colloid cancer, sarcoma of the kidney, horse-shoe kidney, and ascites from other causes. It may be put down to appendicitis because of a lump in the iliac region, or the lump may be regarded as fæcal. It may be ascribed to typhoid fever because of fever and diarrhœa. Early cases are often mistaken for digestive disturbance because of the wasting and large abdomen, but in these there is neither fever nor ascites. The large belly of rickets ought to give rise to no difficulty. Abscess and fistula of the umbilicus are almost always tuberculous, but may be pneumococcal. Examine for tuberculosis elsewhere, especially in the lungs and mediastinal



glands. In doubtful cases some of the recent tests for tuberculosis must be tried.

*The treatment* is general and local, symptomatic and objective. The child must be kept in bed until all definite signs of acute inflammation have subsided. At this stage the treatment is that of acute peritonitis. In very prolonged cases going on well, except for slight fever, the child may be moved out, while in bed or on a couch, into the open air. Open air treatment is of the greatest value, but in the acute stages better results are obtained from careful supervision and nursing, even in large towns, than from sea air or other surroundings which are sometimes supposed to render skilled medical attention and nursing superfluous. Local rest is obtained by bandaging the abdomen and careful diet. The more acute the case the more liquid must be the diet. It may have to be limited to peptonised milk and meat juice. As improvement takes place the diet is increased by the addition of soups and broth, milk-tea, Benger's food, cocoa and gruels made with milk and water, eggs beaten up with milk and water, pounded up fish, meat, mashed potatoes and cream, lightly boiled eggs and bread and butter. Brandy is given in acute stages, and port wine, Burgundy, ale or stout during convalescence. Careful dieting generally stops the vomiting, but, if not, give bismuth, small doses of cocaine or laudanum. Constipation is best treated by diet, cod-liver oil and malt, and castor oil, small doses of calomel or glycerine enemata. Diarrhœa is generally stopped by careful diet, unless there is intestinal ulceration. If troublesome, it must be treated with large doses of bismuth and small doses of opium. Ulceration may be present without diarrhœa. Creosote, guiacol and styracol are useful for tympanites and diarrhœa. I generally prefer to give internally a mixture of cod-liver oil, maltine, and phosphates of iron or hypophosphites ; together with iodoform gr.  $\frac{1}{2}$ -2, or creosote m.  $\frac{1}{2}$ -2, in pill form 3 times a day. Locally, the abdomen is painted with a mixture of belladonna ointment, blue ointment and olive oil (p.a.), night and morning, and a broad flannel binder put on. Hot fomentations and opium are useful for the relief of pain. Under treatment by careful diet, iodoform internally, and local applications, many cases have done extremely well. Most importance must be attached to diet.

*Operative Treatment* is probably of little value unless there is purulent exudation or a possibility of clearing away caseous masses. In the adhesive type it is unnecessary. If there is much effusion of serum, it may do a little good by removing exudates containing toxins and relieving distension. Paracentesis may injure the gut. Local caseous masses often dry up, calcify, and become encapsuled. Even if removed there is almost just as great a probability of a fresh outbreak from disease elsewhere. Operative treatment has shown that the disease is by no means always fatal, while medical treatment proves that operation is usually unnecessary. Purulent effusions are generally localised and treated as chronic abscess. Operation



is also needed for the cure of intestinal obstruction, and may be valuable for small fistulæ.

**Abdominal Cysts and Tumours.**—*Mesenteric Cysts* are of embryonic origin or due to parasites, e.g., hydatid cysts. They do not originate in lymphatic structures. Cysts are spoken of as serous, chylous or sanguineous, according to the nature of their contents. Embryonic cysts are derived from the remains of foetal organs, remnants of the Müllerian and Wolffian ducts and bodies, commonly situated between the layers of the mesentery. Occasionally a cyst is due to dilatation of Meckel's diverticulum, which becomes constricted and attached to the gut by an impervious cord, or to constriction of the ends of an unobliterated urachus. A urachal cyst is not a mesenteric one. In other instances it is derived from a sequestered portion of some retro-peritoneal organ, such as the pancreas; or it may be a dermoid, derived from a sequestered part of the ovary or parovarian body, and containing an imperfect foetus, which may even be alive. Possibly the cyst may arise from a snared-off portion of the gut, or an atypical intestinal diverticulum. Thus, in a girl, aged 5 weeks, a saccular dilatation of the small intestine communicated with the jejunum by a small orifice, 15 ins. from the pylorus. Its walls were composed of normal gut. Had it been entirely cut off it would have developed into a cyst. Multiple diverticula of the large and small intestines have often been reported, and many of them are in the mesentery. Pancreatic diverticula of the small intestine are usually connected with the third part of the duodenum, occasionally the upper part of the jejunum. At the cæcal end is a glandular mass of pancreatic tissue. The pancreas is developed from twin buds or diverticula of the duodenum.

Mesenteric cysts are more common in females than in males, and may occur at any age, e.g., a male, aged 11 weeks (Eve). They are situated between two layers of the mesentery, and occasionally the mesocolon. A prominent fluctuating tumour is found near the umbilicus, and rarely in other situations. It is very mobile, sometimes can be rotated, and easily moved from side to side. A zone of resonance round the tumour and a belt of resonance in front are due to intestine. By pressure on the gut it may give rise to more or less intestinal obstruction and gastro-enteric disturbance. If left alone it may end in death from emaciation, obstruction, rupture or perforation of the gut. The treatment is surgical.

*Pancreatic Cysts* are traumatic or non-traumatic. Coombs and Nash collected 25 traumatic cases of which 3 were under 10 years of age, and 29 non-traumatic of which only their own patient, a girl, 4 years old, was under 15 years. It was probably due to rupture of a small pancreatic cyst into the lesser peritoneal cavity. There are no serious symptoms apart from pressure. Telling and Dobson (1909) reported a case at 11 months of age. There were no symptoms of ill-health, except gradual loss of weight and enlargement of the abdomen. The cyst contained 1½ pints.



Pain is not constant. It is sometimes severe (*Cæliac neuralgia*). The tumour gives rise to dullness, tenderness, and resistance in the epigastrium and left hypochondrium, just below the edge of the ribs. Its contents are alkaline, albuminous, and contain ferments, if connected with the pancreas. Traumatic ones may be merely localised serous effusions. These cysts are treated by incision and drainage.

Tumours of the pancreas are very rare, and usually mistaken for renal tumour. Malcolm (1902) reported a case of fibro-sarcoma in a girl of 4 years.

*Omental Cysts and Tumours.*—Cysts of the great omentum are rare. In a case of Mathews (1905) the contents were sanguineous. The cyst was in the middle line and connected by a pedicle with the transverse colon. Sarcoma may occur and give rise to no definite symptoms. It usually begins on the left side and grows very rapidly. Tuberculous masses are by no means infrequent. Retro-peritoneal Sarcomata are soft and fluctuating and may feel cystic. The gut is situated in front.

*Tumours of the Alimentary Canal.*—Corner and Fairbank (1904) reported a case of sarcoma of the colon in a boy, aged 9 years, causing intussusception. They state that it is the fifth recorded instance of such a result, and the eleventh recorded instance of sarcoma of the colon. In a *resumé* of 174 cases sarcoma was twice as frequent in males, and the round cell variety was the most common. Sarcomata of the ileo-cæcal region and colon are most frequent during the first decade. The distribution of 155 cases was:—œsophagus, 14; stomach, 58; ileo-cæcal, 65; colon, 11; rectum, 7. The symptoms vary with the site; and the course of the disease may resemble tuberculous peritonitis or intestinal obstruction. The prognosis of early operation for sarcoma of the colon is good; 6 were free from recurrence 1-9 years later. Glandular infection is rare, and the secondary growths are usually in the liver or kidneys. Colloid carcinoma has also been reported in the rectum, and generally distributed throughout the peritoneum.

*Multiple Cysts* have been found in the wall of the colon and even more rarely in the ileum. They contain mucin, and are probably due to occlusion of Lieberkühn's ducts. They are situated between the epithelium and serous coat, and may attain the size of cherries.



## CHAPTER XXXII.

### DISEASES OF THE LIVER.

*Jaundice—Acute Yellow Atrophy—Biliary Cirrhosis—Atrophic Cirrhosis—Syphilis—Tuberculosis—Active and Passive Congestion—Abscess—Fatty Liver—Amyloid Liver—Hydatids—Tumours.*

The liver is a most important organ in foetal life. It is formed very early and grows rapidly. By the end of the third or fourth month it nearly fills the abdomen and almost equals half the entire weight of the foetus. At birth its weight is from 4-5 per cent. of the body weight, or 4-5 oz. It is equivalent to the weight of 10 spleens, 9 kidneys, or 7 hearts (Boivard and Nicoll, 1906). In bulk it is greater than that of the 2 lungs combined, and it contains about one-fourth of the total blood in the body. It doubles its weight in 1 year, trebles it in 2 years, and quadruples it in 4 years. It is very susceptible to disease or gives rise to disease, on account of its great size and the relationship which it bears to the foetal circulation. During intra-uterine life the umbilical vein carries arterial blood to the foetus. Entering at the umbilicus it passes along the edge of the suspensory ligament. It gives off branches to the left lobe, the quadrate lobe, and the lobe of Spigelius. At the transverse fissure it divides into two branches; the larger unites with the portal vein and enters the right lobe, while the smaller forms the ductus venosus and joins the left hepatic vein as it enters the inferior vena cava. Hence the maternal blood reaches the vena cava by three different channels. The main bulk passes through the right lobe of the liver and the right hepatic vein; some passes through the left lobe and the left hepatic vein; and the rest joins that of the left hepatic vein, and does not pass through the liver at all. On ligature of the cord the main blood supply of the liver is at once cut off. The umbilical vein is obliterated in 2-5 days, and forms the round ligament, and the ductus venosus is closed in about the same time. The lungs are inflated and exercise their functions. The importance of the liver is diminished, but it still remains relatively larger and heavier than in adult life.

In infants the ribs are more horizontal than in adults, and the liver more exposed. At birth the edge reaches 2-3 ins. below the costal margin, and during the first 3 years of life it can usually be felt an inch below the edge of the ribs. The upper border of dulness is higher than in the adult, and reaches to the lower edge of the fifth rib in the nipple line, the seventh



rib in the mid-axillary line, and the ninth rib behind. The organ may be displaced downward by deformity of the chest, as in rickets ; by fluid or new growths in the thorax ; or from weakness of its ligamentous supports and relaxation of the abdominal muscles in great debility. The right lobe may be so much displaced as to reach the crest of the ilium, leaving the left lobe only in contact with the diaphragm. Hepatoptosis is sometimes marked in acute febrile diseases. Displacement upward is due to fluid or new growth in the abdomen, or excessive tympanites. Transposition is rare. Bile is secreted in the third month of foetal life (Zweifel). Glycogen formation begins in the fifth month. Fat is distributed throughout the lobules.

**Jaundice.**—Although jaundice is not a disease, it is a common and very obvious symptom, indicative of a slight and transient disorder or severe and rapidly fatal disease. Its occurrence in the newborn has already been considered (p. 133). In the present section the simple obstructive or catarrhal jaundice will be considered. Its most usual cause is catarrhal inflammation of the duodenum, with or without catarrh of the stomach, and hence it is often called *Duodenitis* or *Gastro-duodenitis*. The swelling of the mucous membrane blocks the opening of the common duct into the duodenum, or the inflammation extends by continuity up the common duct and into the hepatic and cystic ducts. This is the true catarrhal jaundice of the obstructive variety, the bile passing into the blood because of its inability to escape into the alimentary canal. Obstructive jaundice is occasionally due to blockage of the duct by a plug of mucus or inspissated bile, gallstone or roundworm, and, very rarely, to stricture of the duct or pressure from without. Catarrhal jaundice is common from the date of the eruption of the first teeth up to 6 years of age, and after that is somewhat less frequent. The attack can often be traced to exposure to cold or to food, unsuitable in quality or quantity or undergoing decomposition. It is most common in the spring and autumn, and sometimes occurs in epidemics ; possibly contagious and associated with septic symptoms. Thus, there are probably two classes of cases : (1) Simple catarrhal, and (2) Infectious.

In the simple catarrhal variety the attack is often ushered in by vomiting or diarrhoea. The child loses its appetite, is fretful, languid and drowsy, and in a few days exhibits yellow discolouration of the skin, conjunctivæ and matrix of the nails. Anorexia, nausea, vomiting, epigastric tenderness and pain may persist for some days, if there is catarrh of the stomach. If the catarrh is limited to the duodenum, pain comes on 2 or 3 hours after the ingestion of food, on its passage from the stomach onwards, and there is tenderness in that region. The urine shows the usual changes in colour ; the stools are offensive, constipated or loose, and vary in colour according to the degree of obstruction. If no bile reaches the intestines they are clay-coloured, but if the obstruction is not complete they are more or less yellowish brown. The deficiency in bile prevents the proper digestion



and assimilation of fats. Marked slowing of the pulse is uncommon in children, and itching of the skin is also rare, but it does occasionally occur, and with it a tendency to troublesome urticarial rashes. The liver is almost always enlarged, and may reach as low as the level of the umbilicus. It is sometimes painful and tender. The staining persists for 2 or 3 weeks, and even for as many months without producing any very great effect on nutrition. In prolonged attacks the mental condition is dull, and the child stupid or sleepy. Slight fever is often present in the early stages.

In the infectious or epidemic type the fever lasts for 8-10 days or less. Occasionally there is prodromal malaise. Usually the onset is sudden, with fever, chilliness, vague pains, vomiting, thirst, offensive breath and perhaps delirium. There are pain and tenderness in the epigastrium and over the liver; the liver and spleen are enlarged; the stools are loose or costive, and there is often a trace of albuminuria. Enlarged congested tonsils are not infrequent and may be the source of infection. Jaundice does not necessarily occur. This infectious type is also seen in pneumonia, scarlet fever and septic infections. *Weil's disease*, or *Icterus Gravis*, is a severe type of infectious jaundice, with swelling of the spleen and liver, and nephritis. It is either a toxic or severe jaundice, or due to infection by micro-organisms, frequently the bacillus coli. In a 4 months old infant (Brüning, 1904), who died in 23 days from the onset, the bacillus proteus fluorescens was found in the urine, and the serum agglutinated the typhoid bacillus. This is a common feature of the serum in jaundice. The bacillus was cultivated from various organs, blood and bile. Post mortem examination showed fatty infiltration and degeneration of the liver and, to a less extent, of the heart; purulent inflammation of the kidneys, and swollen lymphoid follicles in the intestines. Fortunately the disease is almost unknown in children. At the onset it simulates simple jaundice and later on may be mistaken for meningitis or typhoid fever.

The *diagnosis* of simple catarrhal jaundice depends upon the history of gastro-intestinal disturbance and some exciting cause. Distension of the gall bladder is evidence of obstruction of the common duct. Other causes of jaundice must be remembered, and excluded as far as possible. It is a complication of many infective diseases.

The *prognosis* is good, if due to simple duodenal catarrh. As soon as the cause ceases to be operative, the blood and urine lose the yellow tint. The re-appearance of normal coloured stools indicates that the obstruction has gone. A sudden return of colour implies sudden detachment of a plug of mucus, inspissated bile or other obstruction in the common duct. Gradual return of colour occurs when the obstruction disappears slowly. The skin resumes its normal colour as soon as the stained epidermis is desquamated. In the rare persistent cases the system appears to accommodate itself to the presence of bile in the tissues for many months. Eventually the obstruction leads to the dilatation of the ducts in the liver, infiltration of the liver cells,



and finally interference with their functions. Changes occur characteristic of a secondary biliary cirrhosis, and the patient dies in a year or two from a kind of toxæmia or blood poisoning. Such a result is not unknown in mild cases, but I have never seen a simple catarrhal jaundice in a child end fatally. Complications are rare. Some children are liable to frequent attacks.

Drug *treatment* is rarely necessary. In catarrhal conditions diet the child carefully, giving bland and easily digested food with a minimum quantity of fat. Fruit is permissible. Constipation is treated by drugs acting on the lower intestinal tract, e.g., saline laxatives, mineral waters, and aloes. It is a moot point whether cholagogues should be given. By increasing the secretion of bile it may be possible to raise the pressure in the ducts and overcome the obstruction. If this fails, the treatment may make the patient worse. Small frequent doses of calomel in my experience are harmless and beneficial, possibly by reducing inflammation, preventing intestinal decomposition, and keeping the bowels gently open. Salol is good for offensive stools. Mild diuretics and diaphoretics aid the elimination of the retained bile. Effervescing mixtures are pleasant. Alkalies are useful by dissolving and liquefying the mucus, and enabling the mucous membranes to digest food more easily. Small doses of nux vomica and dilute hydrochloric acid assist digestion. Emetics are sometimes given in the hope that the act of vomiting may compress the liver and bile ducts sufficiently powerfully to drive out the obstruction. It is experimental treatment, and might cause rupture of the duct, if the obstruction is due to gallstone.

**Acute Yellow Atrophy.**—*Syn.*: *Malignant Jaundice*—*Hæmorrhagic Jaundice*—*Typhoid Icterus*—*Hepatite Maligne*—*Atrophic Aiguë du foie*—*Parenchymatous Hepatitis*.—This disease is very rare in children, and closely resembles phosphorus poisoning. It has been asserted that all cases are due to this cause. Arsenic and antimony produce similar effects upon the liver. Possibly it is due to microbial infection, or to ptomaines or leucomaines acting as poison to the liver. Undoubtedly it is due to a protoplasmic poison which affects the liver chiefly. Leucin and tyrosin are found in the urine, on evaporation to a small bulk and allowing them to crystallize out. It has been recorded as early as the tenth month of life, and about 25 cases under 12 years have been reported. The disease is characterised by acute degeneration of the liver, with the abolition of its functions and death from toxæmia. The symptoms are the same as in adults. It often begins insidiously, like mild catarrhal jaundice, and the temperature remains normal or subnormal, but may rise towards the end. Sometimes the onset is acute with high fever and hepatic pain. The usual symptoms are fever, furred tongue which becomes dry and brown, fœtid breath, sordes on the lips and teeth, nausea and vomiting coffee-ground vomit, tarry stools, jaundice, pain on pressure over the liver, prostration



frequent variable pulse ; marked tendency to hæmorrhages such as epistaxis, purpura, hæmatemesis and melæna ; bile, leucin and tyrosin in the urine ; headache, nervous irritability, somnolence alternating with restlessness, insomnia, delirium, clonic and tonic spasms, convulsions, maniacal symptoms, and coma. The liver may diminish rapidly in size in the second stage, and is often tender. The spleen is enlarged. The pupils often become dilated and react badly.

The diagnosis is impossible in the first stage. Later on it simulates meningitis or typhoid fever. In extreme fatty degeneration the spleen is not enlarged, and leucin and tyrosin are absent. These substances may be absent in mild cases of acute yellow atrophy, and possibly in the most severe ones.

Death commonly ensues within a week. The early stages are of variable duration, but the closing toxæmic stage usually ends fatally in 1-3 days. Bauer (1893) reported a case of recovery, followed by death 3 months later from acute miliary tuberculosis, with marked evidence of regeneration of liver tissue post mortem. No treatment is of any value.

**Biliary Cirrhosis.**—*Syn. : Hanot's Cirrhosis—Hypertrophic Cirrhosis with Jaundice.*—Cirrhotic changes can be produced in the liver by irritants reaching the bile ducts, and setting up cholangitis and pericholangitis, or from chronic obstruction of the hepatic duct or common bile duct, or absence of the ducts. Possibly the irritant is excreted into the smaller bile ducts and sets up cholangitis and proliferation of the surrounding connective tissue cells. Such a result was produced by Hunter as the effect of experimental poisoning by toluyldiamine. Hence the disease may be a primary infection of the minute bile ducts, or an ascending infection from the larger ducts, or due to obstruction.

Hanot's cirrhosis is rare in children. It gives rise to dull pain in the region of the liver, acute attacks of abdominal colic, and slight jaundice which increases with each attack. For some time the health and appetite are unaffected. Gradually the liver and abdomen increase in size, the spleen is enlarged, the urine contains much bile pigment, and the stools are not clay coloured. Fever is slight and may become intermittent. The child wastes and dies with symptoms of cholæmia. Subcutaneous petechial spots, bleeding from the gums and nose, and intestinal hæmorrhage may occur. The fingers and toes may be enlarged, and ascites supervene towards the end. The liver is symmetrically enlarged and smooth ; cuts with difficulty, and has a yellow or dark green hue on section ; and the ducts are pervious. Microscopically there is found a monolobular cirrhosis starting round the bile ducts ; new fibrous tissue in concentric layers round the larger ducts ; and multiplication of bile ducts containing inspissated bile and minute calculi. The disease apparently begins in or around the small bile ducts.



Acute cases may end in a few months ; more usually the jaundice is chronic and lasts for years, perhaps with occasional intermissions. Death results from cholæmia, profuse hæmorrhage, or intercurrent disease.

**Infantile Biliary Cirrhosis** is a disease prevalent in East India, in Calcutta and other large Indian towns, and in Bengal. It is most frequent in Hindoos ; more common among the well-to-do ; and generally begins in the seventh or eighth month of life, sometimes within a few days of birth, and rarely after 3 years. It runs in families, child after child dying from it under the age of 2 years. The onset is insidious, and the liver may be much enlarged before the disease is suspected. It ends fatally in 3-12 months, and occasionally in 2 or 3 weeks. Its causation is uncertain, but it is probably the result of some irritant of gastric origin, e.g., condiments, acting directly on the liver cells or on the lining of the ducts. Syphilis, malaria and alcohol can be excluded. The liver is enlarged, deeply bile stained, and shows marked cirrhosis of the intra- and inter-lobular type, and proliferation of bile ducts. In some parts the cirrhosis is even pericellular. The liver cells undergo degeneration. Similar changes have been produced experimentally by ligature of the common bile duct (Wickham Legg, 1873), or branches of the main duct (Vaughan Harley). Probably, therefore, it is due to obstruction.

The symptoms are languor, fretfulness, sallow skin, a voracious appetite alternating with anorexia, nausea, occasional vomiting, great thirst, constipation and clay coloured stools, and some fever. The liver may extend to the level of the umbilicus or lower, with smooth surface, rounded or prominent edge, and no pain or tenderness. The spleen is enlarged. The sallowness deepens into profound jaundice ; the urine is bile stained ; ascites and puffiness of the hands and feet ensue, and death results from cholæmia, hæmatemesis, melæna or coma. The liver contracts during the progress of the disease. Treatment is entirely symptomatic.

**Congenital Cystic Disease.**—Congenital cystic disease of the liver is usually associated with cystic degeneration of the kidneys, nearly always in a more advanced form, and rarely with cystic degeneration of the lungs and pancreas. It has been found with other congenital anomalies, e.g., hemicephalus and transposition of the viscera (Witzel) ; polydactyly and meningocele (Rolleston) ; polydactyly, encephalocele, hare-lip and malformed external genitals (Couvelaire and Porak). Its pathology is uncertain, but it is probably due to maldevelopment in most cases. It gives rise to no jaundice or other symptoms indicating disease of the liver, although the liver may be so large in the foetus as to interfere with labour, and sometimes reaches to Poupart's ligament. It is compatible with considerable length of life, and may terminate in ascites, œdema and progressive asthenia. Rapid growth is indicated by pain. The liver is enlarged, yellow, firm, and shows white patches. There may be no naked-eye changes. The cysts are scattered throughout as minute white specks in the early



stages, and later on, vary in size from a pea to a cherry, or even larger, and contain no bile. MacMunn (1906) reported a case in a man of 46 whose liver weighed over 12 lbs., and was covered with large transparent cysts, the size of a pea to a Tangerine orange and multilocular. The left lobe was converted entirely into cysts, and the right one almost so. The kidneys and the adrenals were similarly affected. It is said that the coloured fluids injected into the bile duct do not pass into the cystic spaces, yet they are found in the early stages lined by columnar epithelium. A variable amount of uni- and multi-lobular cirrhosis is present. In another type of case in which numerous cystic spaces are found filled with bile-stained debris, there is evidence of prolonged pericholangitis and fibrosis round the bile ducts.

**Atrophic Cirrhosis.**—The typical “hob-nailed” liver may occur in children who have never tasted alcohol. It is probably induced by toxic bodies absorbed from the intestine as a result of gastro-intestinal catarrh, set up by highly stimulating food, alcohol, and even vinegar; or alcohol may act as a direct hepatic irritant. Some cases follow specific fevers. The cirrhosis is of the ordinary multilobular type, an interstitial hepatitis or portal cirrhosis, with secondary fatty degeneration of the liver cells. Palmer Howard (1887) reported 2 cases in one family and collected 61 others; the common age was 9-12 years. It was twice as frequent in boys as in girls. In half the cases no cause could be found except defective diet, only 10 could be ascribed to alcohol and 7 to syphilis.

The early symptoms are those of hepatic incompetence or lithæmia; flatulent dyspepsia, abnormal appetite, headache, debility, languor and lithuria. Morning sickness and attacks of biliousness or diarrhœa may occur. Its course is characterised by digestive disorders, wasting, dryness of the skin, ascites and hæmorrhages. Epistaxis and cutaneous hæmorrhages are common, but hæmatemesis and melæna are less frequent than in adults. Headache and vomiting are common; and jaundice, fever and diarrhœa more frequent than in adults. In a few cases the ordinary signs have been absent, and there has been a marked nervous disturbance with screaming, convulsions and coma; progressive idiocy, paralysis, emaciation and fever; spastic rigidity of the limbs, normal reflexes and loss of speech; high fever and most of the signs of typhoid, or of acute peritonitis, due to perforation. If there is ascites, it is liable to be mistaken for tuberculous or other forms of chronic peritonitis.

A boy, aged 9, under my care died from severe hæmatemesis. At age 3 or 4 he had jaundice for 8 weeks; at 7 years he was in hospital for jaundice and large liver. The terminal illness lasted 4-5 weeks before and 7 weeks after admission to hospital, and was characterised by anæmia, wasting, hæmatemesis, melæna, ascites, irregular fever, and purpura 2 days before death. The liver weighed 32½ oz.; spleen 11½ oz.

The prognosis is extremely bad, especially if there are hæmorrhages or nervous symptoms. Targett has reported a case of a boy, aged 8, who was



apparently in perfect health when killed by accident, and yet had a typical hob-nailed liver. The treatment is essentially the same as in adults.

**Syphilitic Cirrhosis.**—Congenital syphilis produces a patchy or diffuse generalised interstitial hepatitis. It takes the form of an intercellular or unicellular cirrhosis; the individual cells being separated by leucocytes and newly-formed connective tissues. The typical variety in infancy is a diffuse acute infiltration with young cellular connective tissue. Small yellowish points of necrotic liver tissue, areas of anæmic necrosis, are often present. The cells atrophy, and both bile ducts and portal vessels may be obstructed.

The liver is enlarged, usually yellow but varies much in colour, with irregular outline, thickened capsule, and dense consistency. On section it may exhibit isolated, small, greyish white or white spots, which look like miliary tubercles. They are called miliary syphilomata; are composed of small round cells, differing only from tubercles in the absence of the tubercle bacillus; and they undergo necrosis. In the patchy variety the yellowish grey areas are distinguishable by their colour from normal liver.

The diagnosis is based on the hepatic enlargement, in conjunction with other signs of congenital syphilis and enlargement of the spleen. There are no special symptoms and jaundice is rarely marked. Nutrition is generally greatly impaired. Gummata, associated with the cirrhosis, are rare in infancy and uncommon in later childhood. They vary in size, are single or multiple, surrounded by new connective tissue cells, and may break down in the centre. The liver cells are active, and sometimes there are amyloid changes. A polylobular cirrhosis may occur from other causes in syphilitic children.

The prognosis is not very bad, provided that the primary disease is energetically treated. Fatal cases die early. Wollstein (1902) states that 11 out of 14 died under 4 months of age, and the others at 7, 13, and 19 months. Broncho-pneumonia is a common cause of death. Many terminate in complete recovery.

**The Liver in Tuberculosis.**—Miliary tubercles are frequently seen on the surface in miliary tuberculosis. Occasionally a capsular hepatitis, spreading into the lobules, is tuberculous in origin. Rarely tuberculous masses are found. Cirrhotic changes may be secondary to cardiac failure and venous stasis, or due to the tuberculous toxin. Alcohol, syphilis, and malaria must be excluded in these cases, for they are probably due to mixed infections.

**Active Congestion of the Liver.**—Active congestion occurs in malaria, phosphorus poisoning, infectious fevers, in the first stages of suppuration, and from high external temperatures or the habitual ingestion of rich and stimulating foods. It gives rise to chilliness, fever, headache, anorexia, vomiting, constipation, and perhaps mild delirium. The child lies on the right side with the knees drawn up. The liver is enlarged, tender and painful. It must be diagnosed from gastritis and perihepatitis. The pain and tenderness can be relieved by hot fomentations, mustard leaf, leeches,



or cupping. Calomel or grey powder, followed by salines, should be given freely. Free sweating and diuresis must be encouraged, and light diet given. Ammonium chloride is said to be beneficial. The attack is soon over, unless it ends in suppuration.

**Abscess of the Liver** has been recorded in 112 collected cases in young children (Legrand, 1906), and as early as the second year of life. It is due to dysentery, traumatism, appendicitis, migration of roundworms into bile ducts, tubercle, pyæmia, pylephlebitis and umbilical phlebitis, typhoid fever and influenza. Traumatism is relatively a more common cause than in adults, the abscess developing immediately or not for some weeks. In pyæmia the abscesses are small and multiple, and the symptoms those of the primary disease.

Liver abscess gives rise to hectic fever, rigors, prostration, sweating, vomiting, diarrhœa and wasting; jaundice, if there is any pressure on or secondary catarrh of the bile ducts; more or less constant pain, often intense, not necessarily localised over the liver; downward enlargement of the liver, bulging of the ribs, and a tumour if it is situated on the anterior surface and not between the liver and diaphragm. Hiccough may result from irritation of the diaphragm. The inflammation may extend to the thorax, or the abscess may rupture, giving rise to pleurisy, empyema, pneumo-thorax and pneumonia. Or it may rupture through the skin, into the alimentary tract or peritoneal cavity. Metastatic abscesses and endocarditis complicate pyæmic cases. Spontaneous cure has followed rupture through the skin or into the respiratory passages. The abscess may remain quiescent for a time and then grow rapidly.

It must be diagnosed from abscess of the abdominal wall. It cannot be differentiated from subphrenic abscess, which for all practical purposes may be included in this description. Marked local signs are in favour of the abscess being single. An exploratory puncture is necessary, for a quiescent abscess simulates hydatid or new growth. Grumous liver pus is obtained. The prognosis is good, if it is single and treated by incision and drainage. Pyæmic cases are almost invariably fatal, and the multiple abscesses only diagnosed post mortem.

**Passive Congestion** is due to the same causes as in the adult, and produces cardiac cirrhosis or the "*Nutmeg Liver*." It may also follow on asphyxia neonatorum and chronic mediastinitis. The early engorgement is followed by more or less fibrosis and enlargement. Over-distension of the liver, due to backward pressure from rapid heart failure, is indicated by pain and tenderness in the hepatic region, sometimes generalised deep abdominal tenderness, or tenderness over the seventh to the tenth dorsal vertebræ. The enlargement may subside if cardiac compensation is established. If not, it becomes chronic; pain and tenderness increase, and ascites and œdema develop. The superficial abdominal veins over the liver are dilated; jaundice is slight and the skin has a dirty greenish hue, and



gastro-intestinal symptoms ensue. The spleen is large, but contracts a good deal in late stages. Mitral disease is the most common cause.

**Fatty Liver.**—Fat globules are normally present in the liver cells, and in great excess from overfeeding, alcohol, deficient exercise and imperfect aëration of the blood. Extreme fatty infiltration is most common in obese children, who take little exercise. It is apt to be produced in tuberculosis, pneumonia, acute infectious disease and intestinal disorders. It is inconstant in marasmus, rickets and congenital syphilis. Fever leads to fatty changes of the nature of infiltration, or a finely granular degeneration preceded by cloudy swelling. A fatty liver may be present at any period of life, even in utero, and is extremely common under 6 months of age. It gives rise to uniform and painless hepatic enlargement with no jaundice, ascites, or splenic hyperplasia. Its colour varies with the amount of blood it contains, and may resemble that of simple anæmia. Its surface is smooth and shiny, pits on pressure, and has a doughy and greasy feeling. On section it is soft, friable and yellow, and oil globules can be scraped off it with a warm knife. Diet and exercise are the most important factors in its treatment.

**Amyloid Liver.**—Amyloid disease affects the liver, spleen, kidneys, intestines and pancreas. It is due to prolonged suppuration, tuberculosis, and congenital syphilis. It is rare under 10 years of age, and runs the same course as that of adults. The child presents a waxy pallor, suffers from vomiting and diarrhœa, with very offensive stools, and develops œdema and ascites. There is no jaundice; sometimes slight irregular fever; much urine, containing much albumin and hyaline casts. Death results from exhaustion, pleurisy, pneumonia or peritonitis. The ascites is due to the kidney affection, and failure of nutrition and circulation. The abdomen, liver and spleen are much enlarged.

**Hydatid Disease.**—Although the *tænia echinococcus* is common in dogs, hydatid disease is rare in this country. Infection is acquired by swallowing water, lettuce, watercress, etc., contaminated with the fæcal discharges of dogs affected with worm. Many cases in adult life have probably been acquired in childhood. The symptoms are mechanical in origin, and vary with the situation. A case was reported in a child of 5 months by Malcolm (1895); another by Stiles (1904), in a girl, aged 9 years, containing about a gallon of fluid. A thrill and hydatid fremitus were present.

**Tumours.**—Non-parasitic cysts are rare, but have been reported in the foetus and the newborn, possibly a variety of cystic disease. Various cases of congenital tumour of the liver and one or both adrenals are on record. They are generally round-celled sarcomata, sometimes spindle-celled, or described as lympho-sarcomata or lymph-adenomata. Primary carcinoma has also been reported in a few cases, with secondary deposits in various organs, and perhaps of the nature of the multiple adenomata. The true



character of many of these tumours is doubtful because of imperfect examination and report.

Some cases have occurred in utero ; a few have followed injury ; no inherited tendency has been proved. The symptoms include rapid development of the tumour, sometimes the only sign, hepatic pain, enlargement of the abdomen and dilated superficial veins, gastro-intestinal symptoms, tumour, rarely jaundice, no splenic enlargement or ascites, occasionally fever, and œdema. They are fatal in a few days to several months.



## CHAPTER XXXIII.

### THE NOSE AND ACCESSORY SINUSES.

*Congenital Anomalies—Nasal Obstruction—Nasal Discharges—Rhinitis, acute and chronic—Epistaxis—Sinusitis—Osteomyelitis of the upper jaw.*

The nose is an organ of smell and the main channel for respiration. Obstruction exerts a serious influence on development and on the general health. In infancy it interferes with suckling.

**Congenital Anomalies.**—In rare instances the nose is entirely absent, rudimentary, cleft, or extraordinarily flattened. Any part of the nasal passages, especially the choanæ or posterior nares, on one or both sides, may be narrowed or occluded. The occlusion may be membranous or bony. Sometimes it is due to adhesion of the soft palate to the nasopharynx. More anteriorly, obstruction is commonly due to a deflected septum, deformity of the inferior turbinal bones, or a hypertrophic state of the mucous membrane covering these bones, especially at the posterior ends. Rarely there is adhesion of the walls of the nasal fossæ; and membranous occlusion of the exterior nares is a pathological curiosity.

In the newborn the nasal passages and naso-pharynx are very narrow. The adjacent sinuses are small or undeveloped. Congenital maldevelopment of the base of the skull, with a low naso-pharyngeal vault and a high palate, exaggerates the condition. Congenital nasal stenosis is usually associated with narrow nostrils, a high V-shaped palate, a V-shaped dental arch, crowded teeth and mouth-breathing. Schröder (1902) devised a mechanical apparatus for stretching the alveolar arch. It consists of a gold plate and a transverse rod of two parts united by a screw which is slowly tightened. Extreme caution is needed in its application.

**Nasal Obstruction** depends on congenital abnormalities or post-nasal causes, such as various forms of rhinitis, new growths and foreign bodies, enlarged tonsils and adenoid hypertrophy, and occasionally cicatricial obliteration of the nares, usually syphilitic, but known also to have followed diphtheria and noma.

Mouth-breathing causes dryness of the mouth and throat on waking, and a constant undue dryness of the mouth, pharynx and larynx. It predisposes to pharyngitis, laryngitis, bronchitis and tuberculosis. Nasal obstruction is more common among the phthisical. The obstruction also leads to the development of pigeon-breast, indrawn ribs, and retraction of



the membrana tympani, due to catarrh of the Eustachian tube and the middle ear. In cases of deflected septum the retraction is worst on the narrowed side. During sleep the tongue drops back on to the pharynx and induces partial asphyxia. Hence arise morning lassitude, headache, deficient growth, ill health, and possibly enuresis nocturna.

There is great difference of opinion as to the effects on the shape of the jaw. Four types of deformity can be recognised :—

- (1) The arch of the lower jaw is larger than and surrounds that of the upper jaw. The lower jaw is prognathous, and the molar teeth overlap those of the upper jaw. The palate is high.
- (2) The upper jaw is badly developed and atrophic. The curve of the alveolar arch is small, and the upper teeth are consequently on the inner side of the lower ones. The palate is very high, and the turbinal bones are approximated to the septum.
- (3) The sides of the upper jaw are approximated and push forward the incisor teeth, which project and overlap the lower ones.
- (4) The lower jaw is normal, but the mouth cannot be closed (open-bite) because the upper alveolar process, anterior to the molars, deviates upward at a considerable angle. The palate is V-shaped.

Either the malformations are due to adenoids, nasal stenosis or other cause of nasal obstruction, or these conditions are secondary to the deformity, from developmental defects dependent on our advancing civilisation, and comparable with the early decay of the teeth and loss of hair. Undoubtedly adenoid hypertrophy causes a well recognised type of facial contour and expression, but this facies can exist without the presence of adenoids, and adenoids do not invariably produce it. These peculiarities are neither hereditary nor racial in origin. They are the result of impeded nasal respiration and excessive external atmospheric pressure. In mouth-breathing the current of air, passing through the mouth to the lungs, abstracts some from the nasal chambers, rarefies the remainder, and reduces the pressure within them and on their walls. Thus, there is negative pressure within the nasal cavities and positive pressure from outside. In addition, the nasal chambers are imperfectly developed from disuse. According to this explanation deflection of the septum nasi can be produced by unilateral nasal obstruction. Another factor, which aids development, is in abeyance during mouth-breathing. This is the action of the tongue. If the mouth is shut, the tongue exerts lateral pressure on the molar teeth and forces them outwards, widening the palatal arch and preventing overcrowding of the teeth.

Against these views it must be pointed out that palatal deformities are often present without mouth-breathing or nasal obstruction, and may be absent in well marked cases of the latter condition. It might, therefore, be assumed that they are due to a common cause, and that there is no causal relationship.



The association of dolichocephaly and a highly arched palate is quite common, and it is likely that they are both the result of impeded anterior nasal breathing, for this affects all the sinuses connected with the nose. Long continued excess of external atmospheric pressure on young and soft bones will induce narrowing of the face and upper jaw. It is not due to adenoids, and there is no association with them greater than normal.

According to W. B. Parsons (1905) anterior nasal obstruction causes deformity of the maxillæ and posterior obstruction affects the bridge of the nose. The lower jaw is apparently but not really affected. In posterior or naso-pharyngeal obstruction the air pressure in the sinuses is increased, and causes the depression and widening of the bridge of the nose. It is important to recognise that both anterior and posterior nasal obstruction can exist in the same patient, and that the removal of adenoids will not cure the results of anterior obstruction. In post-nasal catarrh the congestion and blockage give rise to symptoms like those of adenoids. It is often associated with crowded teeth, receding chin and a small, narrow naso-pharynx. These are the kind of cases in which there is so-called recurrence of adenoids, i.e., adenoids are present and are removed with a certain amount of temporary benefit, but the signs of nasal obstruction soon recur.

**Nasal Discharge** is the chief indication of nasal mischief. It may be unilateral or bilateral, acute or chronic, serous, sero-purulent or mucopurulent, purulent, blood-stained or offensive. A unilateral discharge is generally due to a foreign body, sometimes to myiasis or new growth, occasionally to affections which produce bilateral mischief, and rarely to disease of the antrum, frontal or ethmoidal sinuses. Bilateral discharges are set up by the various forms of inflammation of the nasal mucosa.

**Acute Coryza**, *acute catarrhal rhinitis* or *simple nasal catarrh*, is common in newborn infants and during the first three years of life. Blockage of the nose by the swollen mucosa leads to inability to suck, mouth-breathing, refusal of food, wasting, fatigue, dyspnœa and sleeplessness. It is commonly due to microbial infection by the influenza bacillus of Pfeiffer, the bacillus *coryzæ segmentosus* (Cautley) or *b. septus*, the micrococcus *catarrhalis* (Kirchner), micrococcus *paratetragenus*, Hoffmann's bacillus, etc. Probably numerous organisms of the influenza group give rise to it. Sometimes it indicates the onset of measles or influenza. The predisposing causes are indoor life, heated rooms, closed windows, excess of clothing, lack of exercise, malnutrition, adenoids and rickets. A deficiency of protein and fat, with excess of carbohydrate food in the diet, is a concomitant cause by lowering vitality and impairing nutrition.

The exciting cause is some organism, perhaps normally an inhabitant of the nose or naso-pharynx, but its virulence is actually or relatively exaggerated by lowered local or general resistance from chill, cold winds, wet, sweating, etc. Even a strong and healthy child, without a trace of adenoids and brought up on the most hygienic principles, may develop an attack



from being out on a cold and windy day and inhaling germ-laden dust, that of wood pavement being particularly irritant to mucous membranes. At all ages the bulk of the cases are due to direct infection from some one already suffering with a "cold in the head." The symptoms and signs are too well known to need description. It frequently starts in the throat, and extends upwards. Fever is often marked in babies at the onset. Extension may take place to the eyes, lachrymal ducts and the various nasal sinuses. Earache and deafness are common. Alimentary disturbance and pulmonary affections arise from the fever, or by direct extension.

Usually the disease runs its course in about a week and clears up completely. Often it leaves behind a certain amount of congestion of the mucosa, a tendency to recurrence, and the development of lymphoid hypertrophy as the result of repeated attacks. Occasionally a *vasomotor* type is seen, in which profuse watery discharge and frequent sneezing persist for a few hours up to a day or so. Nasal catarrh must be regarded seriously in the newborn and in delicate infants, for it is apt to end in fatal bronchitis, atelectasis, or even sudden death.

*Treatment* is more essential in infancy than in later life. Keep the child in bed in a warm room, at 60-70° F., and have the air moistened in dry weather by a bronchitis kettle. Place a pledget of absorbent wool, soaked in 0.001 per cent. adrenalin solution, in each nostril alternately for 2 or 3 minutes. It may be repeated every 3 or 4 hours in bad cases, before feeding. The introduction of a little borated vaseline into the nares induces sneezing and clears away mucus. Apply to the edges of the nose and to the upper lip, to prevent excoriation, cold cream or an ointment of boric acid gr. 20, salicylic acid gr. 3, vaseline oz.  $\frac{1}{2}$ . For crusts use weak white precipitate ointment. Other topical applications, instead of adrenalin, are chloral hydrate gr. 5-10, or menthol gr. 2-5, in olive oil oz.  $\frac{1}{2}$ ; menthol gr. 1, camphor gr. 1, liquid albolene oz. 1. It may be necessary to insert a soft rubber catheter in each nostril to keep the passage open. In the later stages, when the discharge is chronic and purulent, the adrenalin application is followed by a drop of 0.5-1.0 per cent. solution of silver nitrate into each nostril; or by painting the mucosa with a 1-2 per cent. solution, after washing away mucus with an alkaline lotion.

Small doses of aspirin or salicin, mild diaphoretics, and a dose of grey powder or calomel are also needed. The diet must be light and digestible. Strong infants can be starved temporarily, say for 24 hours, and at any rate they need not be pressed to take food. Feeble infants must be fed, by gavage if necessary, and often require alcohol and strychnia. Older children are treated on the same lines. A hot mustard foot-bath, hot diaphoretic drink and Dover's powder may cut short an attack. Inhalations of turpentine, ol. pini sylvestris, ol. eucalypti, formaldehyde and menthol, or menthol and camphor with eucalyptus oil can be used. Protargol, 10 per cent., may be sprayed or painted on the nasal mucosa.



once or twice in the early stages. Borated vaseline, 10-20 per cent., snuffed up into the nose, which is then blown at the end of 10 minutes, is also useful. In blowing the nose, only one nostril should be closed at a time. Snuffs are inadvisable for children, and douching is rarely necessary, though undoubtedly it can be used with great benefit if the child will submit to the treatment. Vaccine treatment is only on trial.

*Nasal lotions* or *Collunaria* are applied by spray, medicine dropper, syringe, irrigation, brush, absorbent wool, or probe and cotton. Plain water, strong antiseptics and astringents must not be used, except silver nitrate as already mentioned. Order lotions in such strength as to render dilution with twice the amount of hot water necessary, for they must be used warm. If ordered at the required strength, the bottle must be stood in warm water until the temperature of the lotion reaches 90° F. Up to the third year the lotion is dropped into the nostrils with the child on its back. Syringing or irrigation may be used for older children, as long as it is not done with sufficient force to drive infection up the Eustachian tubes.

*Syringing*.—Use a glass syringe of  $\frac{1}{2}$  oz. capacity, and cover the nozzle with a piece of soft rubber tubing for insertion into the nostril to prevent injury. Place the child on its back with the head turned to one side. Stand behind the head and syringe through the upper nostril, allowing the fluid to flow out through the lower nostril or the open mouth. Or the child may be in the sitting posture with the head well forward and a basin under the chin. For older children a small rubber ball syringe can be used. The stream is directed along the floor of each nostril alternately, while the child breathes deeply through the mouth. Irrigation is inadvisable for children under 4 years of age, and very little force must be exerted.

The chief nasal lotions are : (1) Normal saline ; (2) Dobell's solution : sod. bicarb. dr. 1, sod. biborat. dr. 1, glyc. ac. carbol. dr. 2 (or Listerine dr. 4), aqua ad oz. 10 ; (3) Equal parts of common salt, bicarbonate and biborate of soda, dr. 1 to the half pint of water ; (4) One part each of sod. bicarb., sod. biborat., pot. chlorat. and 2 parts of white sugar, dr. 1-2 to the half pint of water. As an astringent lotion use sulphocarbolate of zinc, grs. 5 to the ounce, by spray or dropper ; or hazeline 20 drops to the ounce.

*Rhinitis due to Adenoids* causes a chronic, irritating, mucoid discharge, sometimes muco-purulent and sanious, occasionally unilateral. It may be continuous, but more often varies from time to time. Discharge and excoriation of the external nares may be the only signs of adenoids. It is really a simple chronic rhinitis or post-nasal catarrh, a variety of snuffles, often occurring in acute and recurrent attacks. Epistaxis, eczema, impetigo and swelling of the upper lip are not infrequent complications. In its more advanced form the affection involves all the nasal mucosa, especially that of the inferior turbinals, and is known as *Hypertrophic Rhinitis*. This induces chronic catarrh and bilateral nasal stenosis. Salt and water



is used to keep the nose clean. The galvano-cautery is useful, and pendulous masses can be removed by snares. Sometimes it is necessary to excise the inferior turbinals.

*Acute purulent rhinitis of infancy* is set up by infection at birth by gonorrhœal or leucorrhœal discharge. It comes on a day or two after birth with redness, swelling and purulent discharge, and prevents sucking. In later childhood it may be also due to pneumococcal infection or measles. It is treated by alkaline lotions and painting with silver nitrate solution, 1 per cent., or other silver compound.

*Snuffles* is a symptom, but the name is generally used for the rhinitis which occurs in congenital syphilis. It must not be assumed that every baby with snuffles has got syphilis, for it may be due to any of the numerous causes of nasal catarrh. The syphilitic type comes on later in babyhood than the acute purulent rhinitis of infancy, or the acute coryza apt to occur in the newborn. It generally begins in 2 or 3 weeks, and there is a family history or other evidence of syphilis obtainable. The inflammation is sub-acute, chronic and persistent, with muco-purulent discharge, nasal obstruction, thickening of the mucosa and the formation of crusts on the top of raw mucous membrane. It may be acute, and followed by sloughing of the mucosa and necrosis of bone. In later childhood the disease takes the form of a gummatous periostitis and necrosis of cartilage and bone, ulceration and ozæna. Use yellow oxide of mercury ointment, 1 in 8.

*Foreign bodies* are the chief cause of a unilateral discharge, which varies in character more with the nature of the object than the duration of its presence in the nose. At first it merely causes a little mechanical irritation, perhaps none at all if it is a smooth bead or button. Frequently the discharge is chronic, profuse, muco-purulent or purulent, often blood-stained, and excoriates the nares. The foreign body is nearly always in the inferior meatus. A recently inserted smooth one can often be evacuated by exciting sneezing, or getting the child to blow strongly through the nose, with the unblocked nostril compressed. If this fails, apply cocaine as an astringent and anæsthetic, stand behind the child, and push a probe or director gently along the floor of the nose, displacing the foreign body upward. Then, using the margin of the floor as a fulcrum for the instrument, push the object upward and forward, and it will frequently glide forward and out. A general anæsthetic may be needed. Subsequently apply an alkaline mildly disinfectant lotion.

*Myiasis* (maggots) is due to the meat fly, *musca vomitaria*, laying its eggs in a fold of mucous membrane. It sets up acute rhinitis, which may end in ulceration, perforation of the septum and even fœtid atrophic rhinitis. The maggots can be killed by chloroform vapour, or the insertion of a warm oily fluid, which suffocates them by blocking up the pores or stigmata of the respiratory system. They are then syringed out. *Nasal polypus*, mucous or fibrous, is rare. It causes unilateral obstruction and serous



discharge ; sometimes bilateral symptoms and headache, sneezing, reflex cough and asthma. It is removed by snare or forceps. Only about 8 cases of *tuberculous disease* are on record. It may produce a large tumour of granulation tissue, simulating sarcoma, and ulceration and destruction of the cartilaginous septum. It is diagnosed by microscopical examination, and treated by curettage. *Fibrinous* or *membranous rhinitis* is almost always due to diphtheria (q.v.). *Rhinitis caseosa* is rare and generally unilateral. It is probably a sequel of chronic suppuration and retention of pus, with subsequent changes in the pus rendering it caseous, putty-like and foetid. It differs from tuberculous disease in the absence of tubercle bacilli, and from choleostoma in its freedom from cholesterin. *Atrophic rhinitis* is almost unknown under 12 years of age. It has ensued on myiasis and congenital syphilis. Cartilaginous, sarcomatous and other *tumours* are very uncommon.

*General Diagnosis.*—Examination is difficult in the young, because of the small size of the nose and the nervousness of the child. Elevation of the tip of the nose, and lifting the ala upward and outward with a probe often give a good view of the inferior meatus, and enable a diagnosis to be made of foreign body, sinus disease, diphtheria, etc. A mirror and speculum are frequently needed. A history of the insertion of a foreign body, exposure to the infection of acute coryza, influenza, measles or diphtheria, or evidence of congenital syphilis is of much assistance. Chronicity of discharge favours adenoids or syphilis, and a prolonged duration suggests the presence of a foreign body. Thick muco-pus generally means adenoids or syphilis. A blood-stained discharge is most commonly due to syphilis, diphtheria, or foreign body. Pus and blood may be present in tuberculous disease. Pure liquid pus is sometimes due to foreign body, more often to sinusitis. In atrophic rhinitis the nasal cavity is large, the mucosa atrophied, the discharge mucoid and watery, large brownish or greenish-grey crusts are formed, and there is distinct ozæna. In rare instances a profuse watery discharge is due to the escape of cerebrospinal fluid.

*General treatment.*—The prevention of rhinitis depends on general hygiene, judicious hardening, proper diet and clothing, and the avoidance of exposure to infection. The last precaution is of the utmost importance in the newborn, during the first year of life, and for the weak and delicate. Cleanliness is the chief feature in the treatment of all nasal affections. Alkaline lotions are used to dissolve mucus ; mild antiseptics are added if the discharge is offensive ; and any local cause, such as adenoids or a foreign body, must be removed. For suggestions refer to the treatment of acute coryza.

**Epistaxis** is uncommon in infancy. It may occur in the hæmorrhagic disease of the newborn, sepsis or congenital syphilis. The ordinary type of epistaxis takes place from a vein, generally in the mucosa over the



cartilaginous septum or any part of the anterior nares. It is more common in boys than girls. The latter are most subject to it at puberty. The predisposing factors are those which lead to deficient vascular tone and alterations in the coagulability of the blood. A few cases are due to general plethora. Others result from local injury or nasal affections. It may occur at the onset or in the course of fevers, such as typhoid, measles, scarlatina, pneumonia, and the malignant types of all specific fevers; during diphtheria; in blood diseases, e.g., anæmia, leukæmia, purpura, scurvy and hæmophilia; and in the paroxysms of pertussis. The rheumatic diathesis is a predisposing cause. Obstructive and congenital morbus cordis may induce it, through causing passive venous congestion. Stooping and tight collar bands may accentuate or start an attack. Often it follows mental or physical excitement or sudden change in temperature from heat to cold. It is most common in hot muggy weather. A family type, associated with multiple telangiectases of the skin and mucous membranes, not connected with hæmophilia, is also recognised in later life.

Bleeding is the first sign, but it may be preceded by a feeling of fulness in the head. It is unilateral, drop by drop, variable in amount, and rarely enough to cause grave anæmia or death. It may be overlooked through the blood being swallowed.

In simple cases the bleeding is a trivial matter. If recurrent, usually about puberty, it produces the usual signs and effects of loss of blood. At the onset of typhoid fever it is unimportant, but it is a bad sign in late stages of diphtheria, and generally an indication of severity in the course of specific fevers and in blood diseases.

Preventive treatment consists in toning up the system generally by fresh air, cold baths, exercise and general hygiene. Tight bands round the neck must be avoided. The bowels of the plethoric must be kept freely open with salines. In these children no other measures are needed. A glass of milk on waking in the morning sometimes prevents the recurrent attacks which occur in children at puberty.

*Treatment of an attack.*—Compress the nose with the finger and thumb, the patient being in a sitting posture with the arms raised behind the head. Discourage sniffing and nose-blowing. Syringe clot away with tepid water, and make the child breathe deeply through the nose. Reflex constriction of the arteries supplying the mucosa can be induced by a cold key, or similar object, put down the back of the neck, and ice or some cold substance in the mouth. Alternate douching with hot and cold water may be needed. Loosen the clothes round the neck. Always look for the bleeding spot and, if it is seen, touch it with caustic, chromic acid, pure carbolic, or the actual cautery. Mild local astringents, such as diluted lemon juice and tannic acid, and gauze soaked in hydrogen peroxide, 5 volume strength, are often useful. Adrenalin applications are



generally followed by vascular dilatation and recurrence. A simple plug of gauze or wool inserted for an inch within the nostril, and lateral pressure with the finger on the ala, is efficacious. Astringent powders are unsatisfactory, for they induce sneezing. Should all other methods fail, plug the anterior and posterior nares by the insertion of strips of gauze along the floor of the nasal cavity until the nose is completely plugged. Remove it in 24 hours. Syringe with weak carbolic lotion if there is any bleeding, and insert another plug. Drugs internally are of little value. Calcium salts may be tried.

**Acute Sinusitis** is often overlooked and regarded as part of acute rhinitis. Chronic cases attract attention by the discharge of pus from the nose, usually from one side. The pus may be swallowed and give rise to dyspeptic symptoms only. Empyema of the antrum is more common than empyema of the frontal sinus, but both are infrequent. The antrum can be distinguished at birth. It has dental, nasal and orbital walls, and is situated just above the alveolus of the first pre-molar tooth, and a little below the infra-orbital groove. Inflammation may spread from it to the nasal cavity. Even at the age of 15 days it has given rise to marked proptosis of sudden onset and the subsequent discharge of thick, odourless, pneumococcal pus through the nose, the babe soon recovering (Beauvois). In the case of a boy, 9 years of age, the onset was with vomiting, fever, and symptoms suggestive of gastric disturbance. On the third day the temperature was  $104.6^{\circ}$  F., tongue foul, and breath offensive. Considerable discharge took place during the night, with profuse sweating and fall of temperature, rising again in the morning to  $104^{\circ}$  F. The course of the case suggested a general nasal infection, and bilateral otitis media ensued, ending in satisfactory recovery. The diagnosis of sinusitis generally depends on localised pain and a discharge of pus, which can sometimes be actually seen exuding from the antrum and on transillumination. An alkaline antiseptic douche should be used gently and fomentations applied externally. Operation may be needed.

**Osteomyelitis or Osteitis of the Upper Jaw**, sometimes called maxillary and orbital periostitis or phlegmon of the orbit, is often erroneously described as empyema of the antrum. It is said to resemble a similar affection in adults, though in them it is by no means certain that the disease is a primary antral affection. There is strong evidence that it is due to injury or infection, and that it is an osteomyelitis of the superior maxilla. Probably in infants it is sometimes secondary to inflammation of the dental sac. The superior maxilla in infancy is spongy throughout and contains many dental sacs. The antrum is very small, and an empyema is more likely to discharge through the nose than to make its way through the fairly thick bone into the soft tissues of the cheek and a dental sac.

In many instances no cause can be found. Some cases are due to injury at birth by pressure of forceps or on the pubic arch, and others to



infection. Probably the infection starts in the alveolus, extends to the dental sac, and then to the bone. It may begin in the nose or by way of the lachrymal duct, secondary to gonorrhœal conjunctivitis. The spongy bone affords a ready means of spread. The dental sac of the first molar is large and prominent, and likely to be affected early. Rupture leads to extrusion of the tooth and the formation of sinuses.

Brown Kelly (1904) states that out of 15 collected cases, 9 started at 8-21 days after birth, and 6 at 1-9 months. The early origin is strongly suggestive of injury during labour. At the onset there may be no special symptoms, or the child is feverish, cries constantly and may have convulsions. Almost always there are swelling and redness of the eyelids on the affected side, sometimes enough to cause closure of the lids ; occasionally conjunctivitis, chemosis and exophthalmos. The swelling may extend to the naso-labial fold. The cheek is swollen and the hard palate bulges. In 2-3 days an abscess forms below the inner angle of the eye, and eventually bursts externally through the alveolus or into the lachrymal sac. Simultaneously there is a purulent discharge from the nose on the same side, increased by pressure on the sub-orbital swelling. Examination of the mouth shows bulging of the hard palate, swelling of the alveolus, and probably a partially erupted canine or molar tooth whose removal gives free exit to pus. A probe can then be passed into a cavity, which is generally mistaken for the antrum and is really a dental sac, and it may impinge on bare bone. Fluid injected into the cavity may escape through the nose. Sometimes the alveolus is primarily and severely affected.

Gradually the dead bone separates, sequestra are discharged, the secretion dries up, and the sinuses close. The illness may be much prolonged because of the slow separation of dead bone, and may end fatally from inanition or sepsis, with multiple pyæmic abscesses, purulent pleurisy and pericarditis. Quite one-fourth of the patients die. The entire superior maxilla may be necrosed.

It may simulate an alveolar abscess. Usually it starts in the anterior surface of the maxilla, and the nasal discharge appears later or not at all. All cases show pathological changes in the mouth, either a sinus or bulging of the hard palate.

Treatment consists in free evacuation. Make an opening in the alveolus, canine fossa, or where the abscess points, and enlarge any sinus which has formed. Syringe regularly. Guard against the passage of pus into the mouth, to lessen absorption and general infection. Radical operations are unnecessary, usually unsatisfactory and deforming.



## CHAPTER XXXIV.

### LARYNGEAL AFFECTIONS.

*The Larynx—Examination—Croup—Laryngitis Stridulosa—Catarrhal, Membranous and Chronic Laryngitis—Œdema Glottidis—Congenital Laryngeal Stridor—New Growths.*

The infantile larynx is small and yielding. Its superior orifice is bounded by the epiglottis, the arytenoids, and the ary-epiglottic folds. These folds are readily sucked in on deep inspiration, becoming approximated and causing inspiratory stridor. The thyroid cartilage is almost semicircular in transverse section. Up to the fourth year, and notably in the newborn, the cricoid plate is inclined posteriorly. Growth is rapid at puberty.

**Examination.**—For many reasons the examination of the larynx is difficult. Certain anatomical peculiarities are obstructive. The frænum linguæ is short, and up to the age of three years interferes with traction of the tongue. The vertical measurement of the mouth and fauces is relatively very short, so that the uvula disappears behind the base of the tongue. The soft palate is more horizontal than in the adult, and the posterior wall of the pharynx slopes more backward; the angle of the head and spine is less acute. These peculiarities are favourable to Kirstein's method of examination. The epiglottis, instead of being spread out, is curved and folded on itself, so that it forms a protective hood over the vestibulum laryngis, and almost conceals the short vocal cords. Voluntary or reflex spasm of the pharynx or larynx, or the copious secretion of mucus, may render the view impossible. An older child may refuse to open the mouth, resist traction on the tongue, does not readily understand and carry out directions as to breathing and phonation, holds its breath, retches and secretes mucus copiously. With patience and training it is possible to overcome these difficulties.

Lack's method requires depression and traction of the tongue, thus drawing the epiglottis forward. No instrument and no force are needed. The infant is supported in the usual position. The left index finger is placed in the mouth and hooked round the hyoid bone, pulling it forward. The finger acts as a tongue depressor and the knuckle as a gag, while the left thumb under the chin steadies the head. A small mirror is used. The younger the infant, the less is the resistance and the easier the examination. A suitably bent spatula must be used for older children with teeth.



Escat recommends the use of a light tongue depressor, merrythought shaped, terminating in two blunt prongs curved so as to fit into the base of the tongue, one fork lodging in each sinus pyriformis on the side of the laryngeal orifice. It controls the tongue and pulls forward the larynx from the posterior pharyngeal wall. A small mirror, mounted on a short stem, is used to push back the uvula and the posterior pharyngeal wall. It is a "forced" examination. The child must be rolled up in a blanket, and the mouth may have to be forcibly opened. Cocaine is necessary; sometimes a general anæsthetic and a gag.

In Kirstein's method a special autoscope is used. The child's head is drawn over the edge of the table and held by an assistant. A spatula is directed downward to press the tongue forward, and the autoscope is held in the left hand. The head is raised or lowered until the larynx comes into view. Tubular specula, mounted at a right angle on strong handles, are more useful. If the child sits upright with the head thrown far back, such a speculum can be passed without much difficulty or discomfort, after thorough cocainisation; or under anæsthesia in the supine or lateral position. Valuable information can be sometimes obtained by external examination, internal palpation and X-rays.

**Croup** should never be used as the name for a special disease, for it merely indicates a group of symptoms due to several causes. "True" croup is an acute laryngitis of a simple or membranous type, generally diphtheria. "False" or "spasmodic" croup is either laryngospasm or laryngitis stridulosa. The symptoms of croup are hoarseness, inspiratory crowing or stridor, cough, dyspnœa and recession of the soft parts of the chest. In other words it means laryngeal obstruction due to catarrh, with or without muscular spasm, to membranous exudation, or to simple spasm. The cough is husky, dry, suppressed, inefficient and stridulous.

**Laryngitis stridulosa, catarrhal, laryngeal spasm, spasmodic laryngitis, spasmodic or catarrhal croup.**—There is little doubt that this is nothing more than a very mild type of catarrh, with secondary adductor laryngeal spasm, which is especially apt to occur in rachitic infants, and those with a long uvula, enlarged tonsils or adenoids, and a neurotic ancestry. It is infrequent in children unless it has previously occurred in infancy. The common age is 1-4 years, and boys are more liable to it than girls. The usual history is that the child has a slight cold, a little hoarseness or nasal catarrh, perhaps a hollow, barking or slightly metallic cough. It is put to bed, and wakes up in a few hours with cough, dyspnœa, crowing inspiration, restlessness and alarm. In mild cases we find hoarseness, croupy cough, general discomfort, no dyspnœa and perhaps slight fever. In severe ones the child sits up in bed in a state of excitement, terror and urgent dyspnœa, struggling for breath. There is loud inspiratory stridor, great recession, hoarseness, stridulous hoarse or metallic cough, laboured and slow breathing, rapid pulse, and a temperature of 99-101° F. Slight



cyanosis, progressing to asphyxia, may be present, with profuse sweating, prostration and the appearance of impending death. The attack lasts from  $\frac{1}{2}$ -3 hours, ends suddenly or subsides slowly, and may recur on the next two or three nights, and every few weeks or at longer intervals. During the day the child is quite well or presents a little hoarseness and cough, and perhaps stridor.

Occasionally the child goes to bed apparently quite well. Sometimes the voice is clear, although stridor is marked. Slight catarrhal sounds may be heard in the chest on the following day. Some attacks last only a few minutes. Although the attacks may be very alarming they are rarely, if ever, fatal. The parents must be warned that they are liable to recur, especially after indiscretions in diet and exposure to cold. A cold bedroom, full of fog, is very likely to induce an attack. Few cases occur after the seventh year.

The treatment is essentially that appropriate for simple laryngitis, combined with that for laryngospasm. For immediate relief rely on hot applications to the larynx, a hot mustard bath, an emetic of vin. ipecac. dr. 1 or pulv. ipecac. gr. 5 half hourly until vomiting is induced, and inhalations of steam. Inhalations of amyl nitrite or a few whiffs of chloroform are more efficacious. Whitla recommends vin. ipecac., vin. antimon., syr. scillæ aa m. 10, every  $\frac{1}{4}$  hour. As attacks are variable in duration and sometimes last only a few minutes it is difficult to estimate the value of the medicine. A dose of bromide, chloral or phenazone at bedtime acts as a preventive. Between the attacks attend to the general health and search for the exciting cause. A warm dry climate is the most suitable. Arsenic, cod-liver oil and belladonna are often useful.

**Catarrhal Laryngitis** varies much in severity. It may occur at any age but is quite infrequent after the fourth year of life, for the child is hardier and less susceptible. The common predisposing causes are rickets, bad hygienic surroundings and confinement in hot, ill-ventilated rooms. An attack is induced by the conditions liable to set up catarrh of the respiratory mucosa, e.g., damp, cold air, irritant vapours, steam, scalds of the mouth, caustics, dust and microbial infections. It is frequent in measles and less common in pertussis, influenza, typhoid fever, scarlatina and other infections.

The onset is more or less acute. After a scald the symptoms come on in a few hours or almost at once. In infancy the symptoms are severe because of the small and yielding larynx. The œdema of the mucous membrane of the arytenoids, upper orifice of the larynx or in the subglottic region, produces serious obstruction in a few hours. A mild attack may pass into a severe one. The main sign is the inspiratory difficulty in breathing, with no intermission and often considerable fever. The temperature ranges from 101-105° F. Sometimes the symptoms are limited to hoarseness, partial or complete loss of voice, a hard cough



which is worst at night, pain and soreness over the larynx and very slight malaise. Such attacks are seen in older children. In the more serious and the infantile type all the signs of laryngeal obstruction are present. The cough is constant, harsh, barking or stridulous. Dyspnœa is inspiratory, mainly paroxysmal, and exaggerated at night. As the case increases in severity, the pulse and breathing become more frequent and the temperature higher. Restlessness is excessive and the child throws itself all over the bed, the colour turns more and more livid, sweat stands out on the forehead, recession of the chest becomes extreme, all the accessory respiratory muscles are brought into play, stridor is loud, and on listening to the chest little or no vesicular breathing is heard. Dyspnœa, cyanosis, pallor and prostration may be as severe as in diphtheria. Unless relief is obtained, death ensues from collapse, cardiac failure, an attack of suffocation and cyanosis, or asphyxial convulsions.

The *duration* varies from 1-6 weeks. Severe symptoms subside in 2-3 days; the child becomes more comfortable and falls asleep as the respiratory obstruction abates. In rare instances the inspiratory dyspnœa persists for weeks without any paroxysmal attacks. The chief *complications* are spasm of the glottis, œdema of the glottis, collapse of the lungs and convulsions. Catarrh of the trachea and bronchi is common and liable to end in broncho-pneumonia. Recurrence is not infrequent.

It is often impossible to be certain that the case is not diphtheria. Chief stress must be placed on the absence of signs of this disease in the nose or fauces. Laryngitis is generally rapid in onset and not accompanied by constitutional disturbance, beyond that due to the dyspnœa. In older children a laryngoscopic examination may settle the diagnosis. The laryngitis at the onset of measles is often extremely acute in onset and development, subsiding with the appearance of the rash. The presence of buccal spots must be sought for. Laryngospasm and congenital laryngeal stridor are unlikely to be mistaken for this affection.

The *prognosis* depends on the age and strength of the child, the degree of rickets, the cause and severity of the attack, and the treatment. The younger the child, the worse is the outlook. The glottic œdema, so often present, is almost invariably fatal in the newborn. Even in the mildest cases there is always a liability to fatal spasm. In measles the child is generally out of danger from the laryngitis as soon as the rash appears. Tracheotomy affords immediate relief, but increases the gravity of the prognosis, for death may result from profuse bronchial secretion and cyanosis, pulmonary collapse and broncho-pneumonia, or infection of the wound. The only sequel of an ordinary attack, untreated by operation, is a certain degree of hoarseness or huskiness and stridor for about 2-3 weeks, and in rare cases persistent throughout life.

*Treatment.*—The mildest cases must be treated with care and watched, because of the risk of spasm and œdema, and the sudden development of a



more severe type. The child must be kept in bed, in a warm, well-ventilated room, fed on light diet, and given a calomel or saline purge. Apply to the throat frequent hot light compresses or a mustard leaf. An ice bag is sometimes more efficacious in severe cases and in acute œdema due to scalds. A tent or a half-tent should be rigged up and a bronchitis kettle used, with eucalyptus, tr. benzoin co., creosote or turpentine added to the water. These drugs may be given by inhalation at 100-120° F., using an inhaler with a large face piece. An emetic may be given to a strong lusty child and chloroform used for the relief of spasm, as in the treatment of laryngitis stridulosa. Preparations of antimony and ipecacuanha are often given in frequent small doses, but on the whole, I prefer a diaphoretic and diuretic mixture. If the skin and kidneys act freely the symptoms are usually relieved. Bleeding is rarely advisable. For high fever, great respiratory distress and objections to operation, a couple of leeches can be applied above the sternum. Should the above treatment fail, recourse must be had to intubation or tracheotomy. In all doubtful severe cases it is advisable to give a dose of antitoxin. If the child has been in bed for at least an hour and grave obstruction shows no sign of yielding to treatment, but is rather getting worse, operate. The main indications are increase in the fever, frequency of the pulse and breathing, restlessness, stridor, recession, lividity, pallor and prostration. Should there be strong evidence in favour of a non-diphtheritic type, tracheotomy may be postponed as long as possible, provided the doctor is on the spot and able to open the trachea at a moment's notice in case of sudden spasm. Intubation is satisfactory, if it can be carried out, and leaves no scar. The method is described in the chapter on diphtheria. Failing expertness in intubating, it is better to rely on tracheotomy, although the subsequent risks are greater. Tracheotomy must be avoided, if possible, in girls who may have to wear low-necked dresses.

In cases treated without operation the treatment by bed, fomentations and inhalations must be continued, until practically convalescent. Subsequently attend to preventive measures. Guard against chill, damp beds and unwarmed rooms. Sponge the neck daily with cold water. Remove adenoids and large tonsils, if present. Attend to the general health. Fresh air and cold bathing are most beneficial.

**Membranous Laryngitis** is commonly diphtheria and is described under that head. Occasionally it is due to the pneumococcus or streptococcus, or to scalds and caustics. In primary laryngeal diphtheria the diagnosis is difficult, because of the slow absorption of toxins from the mucous membrane of the larynx and the preponderance of the obstructive laryngeal symptoms over the constitutional ones. The signs of a membranous laryngitis are like those of the catarrhal type, but the onset is more gradual, the course more steadily progressive and the fever less marked. Hoarseness is succeeded by loss of voice. Dyspnœa steadily increases, and becomes



inspiratory and expiratory in character ; and all the features of obstruction are added to an increasing pallor, lividity and prostration. Unless the obstruction is relieved, the restlessness passes into a state of semi-stupor, coma, and death from exhaustion, sometimes preceded by fits. Towards the end the temperature may rise rapidly or fall below normal.

Some cases run a milder and more chronic course, the symptoms being merely indicative of laryngeal catarrh, with little fever or dyspnœa. Even in infants I have known such attacks to last for weeks, the child recovering after coughing up membrane or after tracheotomy. Generally in infants death results in a day or two. More prolonged cases are complicated by extension downwards of the membrane, and tree-like casts of the whole bronchial system may be coughed up, as in fibrinous bronchitis. Broncho-pneumonia is a common fatal termination.

The membranous type has to be diagnosed from other forms of laryngitis, foreign bodies in the larynx, capillary bronchitis and broncho-pneumonia, and from retro-pharyngeal abscess. The treatment is that of diphtheria. Calomel fumigations have been used, when antitoxin is not available and for cases due to other organisms. The child lies down in a closed tent and from 10-15 grs. are vapourised every 1-3 hours. The danger of fire must be guarded against.

**Chronic Laryngitis** sometimes follows the acute form, especially when due to measles or diphtheria ; may be due to a papilloma or foreign body ; sometimes depends on bad hygiene ; and in a mild form is the result of general congestion of the fauces, secondary to adenoids or mouth-breathing. It is treated on general principles, and, in older children, by astringent sprays, inhalations or insufflations, e.g., ac. tannici gr. 20, glycerini m. 90, aquae ad. oz. 1. The *syphilitic* form is described in the chapter on congenital syphilis. *Tuberculous* laryngitis is practically unknown before the fourth year and rare at a later age. It has occurred although the lungs were healthy, but is generally secondary to pulmonary tuberculosis. The signs are hoarseness, cough, and muco-purulent sputa in which tubercle bacilli may be found. It differs from the syphilitic type in the greater local sensitiveness, more discomfort or pain on cough and swallowing, greater swelling of the arytenoids and less destruction of the epiglottis. Unless there are definite lung signs it is usually mistaken for syphilitic ulceration. Laryngoscopic examination in the early stages shows little difference, but local tubercles may be seen and the process is more superficial. The prognosis is almost hopeless, and the treatment the same as in adults.

**Œdema Glottidis** is either serous or inflammatory. It is induced by the stings of insects, inhalations of steam or irritants, foreign bodies, injury to the larynx, laryngitis, perichondritis, faucial affections and post-pharyngeal abscess. Or it depends on nephritis, morbus cordis, venous congestion, the effects of potassium iodide, and, in rare instances, angioneurotic œdema. In all cases the ary-epiglottic folds are infiltrated with effusion



and form diffuse swellings, which block the upper orifice of the larynx. The arytenoids and epiglottis may be similarly swollen. In serous œdema the swelling is pale red in colour; in the inflammatory type it is brighter red, more diffuse and involves the adjacent structures.

Symptoms develop with great rapidity and may prove fatal in a few hours. Sometimes the onset is preceded by tickling in the throat, pain on swallowing, alteration in the voice and fever. The swelling gives rise to great inspiratory dyspnœa, stridor and attacks of suffocation. Expiration is stridulous or unaffected. Pallor, cyanosis, restlessness, rise of temperature, and increased frequency of pulse and respiration are present. Simple œdema rarely causes the pain, dysphagia, hoarseness and cough of the inflammatory affection. On digital and laryngoscopic examination the swellings are found in approximation near the base of the tongue. They can be seen on pulling the tongue forward. The affection may be mistaken for diphtheria, erysipelas of the throat or asthma.

For true œdema apply ice externally, give ice to suck, and scarify or make multiple local punctures. Inject pilocarpin, if it is of renal origin. For inflammatory cases adopt like measures, and leeches or tracheotomy if necessary. Attend to the cause.

**Congenital Laryngeal Stridor** is an abnormality of respiration which may be present at birth but is more usually noticed, or comes on suddenly, during the first few days of life. In all other respects the child seems healthy and the naso-pharynx is normal. It varies in intensity from a purring, crowing, cackling, grunting or harsh croaking inspiratory sound up to distinct stridor. In severe cases a slight noise is heard on expiration. The maximum intensity is at the end of inspiration and during excitement it may terminate in a "crow" like that of laryngospasm. It is almost always absent during sleep. In one case under my care it persisted to a slight extent. It is worst in the dorsal position; increased by catarrh and any cause of deeper breathing; and there is often a certain amount of recession of the soft parts at the root of the neck, the epigastrium and even the lower ribs. As a rule there is neither dyspnœa nor cyanosis; possibly dyspnœa indicates a complication. In one instance I noted frequent attacks of urgent dyspnœa and inspiratory stridor for which no cause could be found. Vocal utterance is clear, crying and cough are unaffected, and the vocal cords are normal.

*Pathology.*—In a fatal case of my own, and in 5 out of 8 collected cases the anatomical state was practically identical; so too in cases examined during life. The epiglottis is abnormally long and folded longitudinally on itself, so that its edges are approximated, leaving a narrow, median chink-like slit. It curves backwards over the larynx and the ary-epiglottic folds are closely approximated, converting the upper orifice into a mere chink. The cartilages of Santorini may touch each other and the opening of the larynx may be smaller than normal.



The infant's larynx is normally soft and collapsible, the epiglottis gutter-shaped, and the ary-epiglottic folds are drawn together during inspiration and narrow the orifice. The sucking in of these folds during inspiration has been observed by Dundas Grant in a 3-year old boy with inspiratory stridor since birth. For these reasons J. Thomson and Logan Turner have argued that the stridor is not due to the anatomical defect and that it is simply an exaggeration of the normal state, dependent on defective coordination of the respiratory movements, a stammering of the respiratory muscles, the sound being due to imperfect action of the abductors and aided by the soft yielding character of the structures forming the superior orifice of the larynx. Similar stridor may occur during recovery from chloroform narcosis, during excitement and on sudden deep inspiration. Hence, the presence of a congenital deformity is not essential to its production. On the other hand there is no evidence of abnormal breathing from this supposed incoordination ; and during anæsthesia of these patients, breathing is regular and stridor very marked. Furthermore, it is a continuous stridor, sometimes persists during sleep, has been noted immediately after birth (Variot), and is unassociated with other evidence of incoordination ; and definite anatomical peculiarities have been found in the third month of life (Refslund).

By reason of the anatomical findings it has been ascribed to congenital deformity of the superior laryngeal aperture *plus* flaccidity (Sutherland and Lack) ; malformation of the larynx ; malformation of the epiglottis and superior opening of the larynx ; valvular action of the superior laryngeal orifice on inspiration ; or flabbiness of the vocal cords. Possibly more than one factor is concerned in its production. Post-mortem evidence strongly supports the anatomical theory and until typical cases are found, which present no such changes after death, we must regard the incoordination theory with suspicion.

Thomson and Turner, in 5 children from 7-9 years of age, found the anatomical condition persistent long after the stridor had ceased. The subsidence of the stridor is probably the result of increase in size of the larynx. Cases in which the stridor begins and ceases suddenly may be due to incoordination, perhaps affecting the vocal cords and not the upper laryngeal orifice, the rima glottidis remaining closed when it should open for inspiration : cf. " blue fits " and " crowing " due to temper in neurotic children. Stridor has been ascribed also to a lax epiglottis (Dundas Grant), vibration of a flap of loose mucous membrane on the summit of the arytenoids (Brown Kelly), and vibration of soft structures on the posterior wall of the larynx, the arytenoids and folds being drawn downward (Paterson).

*Prognosis.*—About ten fatal cases are on record but death is due to intercurrent disease, possibly caused by spasm in one. The stridor may last for several years. As a rule it increases in loudness for a few months and terminates before the end of the first or second year of life, perhaps quite



suddenly. The malformation persists for years, though the stridor ceases, but may recur under sudden emotion.

The *diagnosis* is comparatively easy. It must not be confused with the noisy breathing of laryngitis, palatal palsy or macroglossia. Laryngospasm does not occur before the third month of life. Adenoids and other forms of nasal obstruction are the main cause of error. The laryngeal spasm set up by adenoids increases during sleep, during feeding and on closure of the mouth, ceases under chloroform, and may be associated with severe suffocative attacks. The stridor due to pressure on the trachea is usually expiratory and associated with more dyspnœa, and is rarely congenital. *Respiratory* or *laryngeal spasm* sometimes occurs in the newborn. An attack of crying is succeeded by crowing inspiration, apnœa and lividity. The initial crow may be absent. These attacks may prove fatal, temporary, or persist in a mild form for some months. They are due to glottic spasm or of the nature of mild laryngitis stridulosa.

*Treatment* is of little value. The child must be protected from spasm, laryngitis and bronchitis. Intubation is too difficult to be of use. Possibly tracheotomy may be required if the anatomical state is extreme.

**New Growths.**—Sarcoma is rare. Papillomata are not uncommon. They are more frequent in boys than girls. About 25 per cent. are congenital. They are most common in the third year of life, and may arise or develop rapidly after laryngeal catarrh. They are single or multiple, variable in size, pedunculated or sessile, and usually on the true vocal cords. Sometimes they are found in the trachea, immediately below the larynx, and may set up tracheal stenosis. They give rise to the symptoms of chronic laryngitis, viz., hoarseness, spasmodic cough, perhaps loss of voice, and sometimes attacks of glottic spasm. Hoarseness and dyspnœa may date from birth. Later on, suffocative attacks and dyspnœa are due to obstruction and stenosis.

Papilloma must be suspected in all cases of chronic laryngitis, and a laryngoscopic examination made. It may disappear spontaneously, be coughed up or cause sudden death from spasm.

*Symmetrical nodules* have been found on the cords in cases of hoarseness, sometimes congenital. They are possibly due to the non-absorption of the embryonic web between the anterior thirds of the cords. Unilateral ones are more common and cause huskiness. They should be left alone for they may disappear at puberty or cause no trouble.

A papilloma can be removed by the endo-laryngeal method, if the child is tractable and the surgeon prepared for tracheotomy in case of necessity. Brüning's speculum is the best instrument. Either forceps or a wire snare is used. Tracheotomy alone is sometimes followed by complete disappearance of the growth and recovery of the voice. If it fails, the growth is removed by the above method or, as a last resort, by thyrotomy



or laryngo-fissure and curettage. This greatly endangers the integrity of the voice. Operations may be followed by bronchitis and broncho-pneumonia. Recurrence is common. Bronner recommends the use of 1 per cent. formalin spray as a preventive. In view of the fact that many disappear without operative interference, it should not be too lightly undertaken. Papillomata often seems to grow more rapidly after operation.



## CHAPTER XXXV.

### THE RESPIRATORY SYSTEM.

*The Thorax—The Lungs and Respiration—Respiratory Symptoms—Atelectasis—Hypostatic congestion—The Trachea—Foreign bodies in the air-passages—Gangrene—Abscess—New growths—Emphysema.*

At birth the chest is almost circular. Towards the end of the first year the transverse diameter exceeds the antero-posterior, and by the tenth year bears the proportion of 3-2. The permanent shape is almost attained by the fifth year. Measurements of the chest are of little value as a standard. The circumference at the nipple level is about 13 ins. at birth, 17 at 1 year, 19 at 2 years, 21 at 5 years and 24 at 10 years of age. Relatively to the spine the sternum is a vertebra higher than in the adult. The ribs are nearly horizontal and the epigastric angle very obtuse.

The muscles are weak, the ribs pliable, and the whole chest wall soft and yielding. This flexibility allows considerable alterations in shape from respiratory obstruction and atmospheric pressure. It also affects the expansion of the lungs in infants kept lying on their backs and may partially explain the better results of private practice. In hospital cases more or less collapse of the posterior parts of the lower lobes is often found after death. The weight of the lungs at birth is about 2 oz., 4-5 oz. at 1 year, and 10 oz. at 7 years of age.

*Examination of the chest.*—The chest must be carefully inspected in a good light for abnormalities, irregularities in shape and uneven expansion. Measurements are taken by a tape measure, callipers and a crytometer at the level of the junction of the fifth rib and cartilage. A small child has a small chest. An abnormally *small chest* is due to naso-pharyngeal obstruction or insufficient air and exercise. The intercostal spaces are small, the ribs unduly oblique and the lower ones turned inwards. The back is straight, the abdomen retracted, and there is a tendency to bring the shoulders forward. Respiratory obstruction must be cured and deep breathing taught in these cases. The outlook is good. Respiratory obstruction has a marked effect in rachitic infants, producing the *rachitic chest* (pp. 181, 185). If the obstruction is marked, this form of chest may be present at an early age, before the development of rickets and independently of this disease. The deformity may entirely disappear in the course of growth. *Pigeon-breast* is apt to occur from debility and uncomplicated respiratory obstruction.



Unilateral flattening is due to chronic tuberculous lung disease, collapse, imperfect expansion of the lung after pleural effusion, or fibroid contraction of the lung. In lateral curvature of the spine the chest is prominent on one side and flattened on the other, and there may be a groove above the level of the liver or spleen equivalent to half a Harrison's sulcus. Unilateral bulging is seen in pleural effusions, lateral curvature of the spine and intra-thoracic tumours. In slight effusions the affected side may be relatively small, because of over-expansion of the opposite lung. Local bulging is caused by cardiac hypertrophy, pericardial effusion, pointing empyema, congenital malformation, greenstick fracture of the ribs, or local affections of the chest wall. Bilateral over-expansion is seen in emphysema, chronic bronchitis and asthma.

Signs obtained by percussion of the back below the middle third of the scapula can be disregarded, for the diaphragm reaches this level in the very young and abnormalities below it require very great experience for an accurate estimation of their value. Note the sense of resistance; it is much increased over consolidated lung and to a greater degree over effusions. The percussion note on the right side above the middle of the scapula is tympanitic in character because of the nearness of the bronchus to the surface. For further details refer to Chapter I, pp. 5-6.

**The Lungs and Respiration.**—The foetal lungs are lobulated, dark red, firm, solid, airless and sink in water. They contain much connective tissue in the form of septa. The *blood* is carried to them by the pulmonary and bronchial arteries. The latter vessels supply the bronchial glands, air passages, large arteries, interlobular tissues and pleura. At birth the placental circulation is cut off and the blood is deprived of its supply of oxygen. The lack of oxygen and the cutaneous stimulation of the peripheral nerves excite the respiratory centre, and cause respiratory movements and expansion of the lungs. At first the breathing is chiefly thoracic and air does not penetrate all the alveoli. Much of the lung remains unexpanded for hours and often expansion is only complete at the end of the second day. Thus there may be partial atelectasis, an important point forensically. The anterior edges of the upper lobes are first expanded, then the upper lobe as a whole, and finally the posterior portions of the lower lobes. The presence of mucus, etc., in the tubes causes patchy and irregular expansion. Atelectasis has been already described (p. 117). During the first few days the interchange of gases is feeble but it soon becomes relatively greater than in adults. The average tidal air amounts to 35 c.c., and may attain a maximum of 120 c.c. *Lymphatics* are situated in the alveolar walls between the capillaries, opening into perivascular lymph spaces, and in the walls of the bronchi. They enter the bronchial glands.

The *respiration rate* is 40-45 per minute in the newborn, 25-30 at 6 months, 24 at 2 20 at 3, and 18 at 7 years of age. A chart of the rate is of much more value than a single estimation. It increases to a greater



extent and is of less serious significance, the younger the child. It is decreased in sleep; increased by crying, excitement and exertion; often irregular up to the third year; diaphragmatic in nurslings and abdominal up to the seventh year. It is normally high in severe rickets, because of deficient lung expansion, and is modified in disease by the presence of rickets. It varies with the temperature, obstruction to the entry of air, the amount of lung involved, and is affected by rapid involvement of lung, pleural pain, weak respiratory muscles and yielding ribs. If the breath is held for long there is no serious lung trouble. Compared with adults there is more oxygen absorbed and more  $\text{CO}_2$  eliminated in proportion to body weight and less in proportion to body surface.

**General Symptoms.**—Apart from changes in respiration, cough, expectoration and hæmoptysis are the chief symptoms of pulmonary disease. *Cough* is useful or useless. Its object is to get rid of normal or abnormal secretions from the fauces and air passages, or it may simply result from reflex irritation of the vagus nerve. A normal reflex can produce or maintain a cough, if there is undue irritability of the nervous system. The chief causes are pharyngeal affections, laryngeal and respiratory disorders, and reflex irritation.

*The Physiological Cough of Infancy.*—Sometimes a baby begins to cough a few days after birth, usually on the second or third day, and continues to do so for even a year. This cough is rare after bottle-feeding; most common in well-nourished, breast-fed babies after the first nursing of the day. It is often associated with regurgitation, a sign of over-distension of the stomach. It is a reflex stomach cough and assists in driving the food through the pylorus. Reduce the duration of each nursing, if necessary.

*Nocturnal or Spasmodic Night Cough* comes on in young children about the middle of the night or shortly after being put to bed. It is spasmodic, somewhat like whooping cough, lasts a long time, and often causes nausea, retching and vomiting. It is almost always due to adenoids and post-nasal catarrh; sometimes to large tonsils or elongated uvula; occasionally to post-pharyngeal abscess, and the pressure of enlarged glands or the abscess of Pott's disease on the trachea. Rarely it is of purely nervous origin or due to the reflex irritation of intestinal parasites. Its spasmodic character is caused by mucus dropping into the larynx and collecting in the region of the arytenoids, setting up glottic spasm and attacks of suffocation. Or an elongated uvula drops back when the child lies down. A cough from this cause comes on shortly after going to bed; that of post-nasal catarrh comes on later and occurs by day also.

*Reflex Cough.*—A knowledge of the branches and distribution of the vagus enables us to account for many troublesome coughs. Wax, foreign bodies and catarrh of the external auditory meatus irritate the auricular branch (ear cough). The pharyngeal branches are stimulated by affections



of the throat, a long uvula, irritant dusts and vapours, changes of temperature, hairs, fluff, fish bones, etc., and possibly pertussis. The superior laryngeal supplies the larynx, epiglottis and base of the tongue. The tracheal branches are stimulated by pressure on the trachea and inflammatory affections, while those to the lungs and pleura are excited by various affections of these organs and passive cardiac congestion. There is no clear evidence of a reflex cardiac cough, but a hard and painful one is sometimes noted in pericarditis. "Stomach cough" is harsh, short and dry. It arises from digestive troubles and disappears when the stomachic state gets well.

*Hysterical Cough* is a peculiar barking cough at about puberty, more common in girls. The child is often said to "bark like a dog." Sir William Jenner held that a "loud barking cough in boys, unrelieved by treatment, is always connected with masturbation." The connection is doubtful, though the two conditions may co-exist. Sometimes it is due to pharyngeal causes or dentition. The child is usually neurotic or of neurotic ancestry; often overfed and allowed too much alcohol; and shows other signs of neurosis. The cough is rhythmical, more or less regular or paroxysmal, harsh and unmelodious. It may simulate an ordinary cough or be curiously monotonous. There is no pain or expectoration. It ceases during sleep and is increased by emotion. Its onset and termination are sudden or gradual, and relapse is frequent.

Consideration of the above varieties of cough shows that it is by no means a constant proof of lung troubles. In quite half the cases it depends on throat affections. Some coughs are due to habit. The nervous and reflex types do not affect the health. Sources of reflex irritation may be present and yet not the actual basis of the cough. The treatment is essentially that of the cause.

*Hæmoptysis* is quite uncommon under 6 or 7 years of age. Even if there is bleeding, the young child rarely expectorates. It is secondary to pulmonary phthisis, gangrene, infarction, or the rupture of a caseous gland into the trachea or bronchus. In malignant forms of specific fevers and blood states, such as hæmophilia, purpura, etc., it is primary. A fatal case in a boy, aged 3 years, was caused by perforation of the right bronchus and the branch of the right pulmonary artery to the upper lobe by a suppurating caseous gland. In a boy, aged 4, cavitation of a gland at the root of the right lung communicated with a bronchus. In the cavity were three branches of the pulmonary artery, one of which presented a fusiform aneurysmal enlargement and had ruptured.

Post-mortem examination generally shows tuberculous arteritis, softening and perforation, sometimes preceded by aneurysmal dilatation; perforation of the trachea or bronchus; or excavation of the lung, primary or secondary to a tuberculous gland. Expectoration may be tinged with blood for a day or two before the fatal bleeding.



*Expectoration* affords little information, for children swallow the sputum. It can be obtained by making the child cough and collecting the sputum from the fauces on a swab of wool, or by lavage of the stomach in the morning before food.

**Collapse** or *acquired atelectasis* is an effect of obstruction of the air passages by secretions and foreign bodies. Rickets, marasmus and debility are the main predisposing causes. It depends partly on feeble inspiration, and is frequent in all forms of bronchitis and broncho-pneumonia. Powerful respiratory movements in these diseases are more likely to produce emphysema. *Compression collapse* is seen in pleural and pericardial effusions, great cardiac hypertrophy, deformity of the chest sufficient to prevent complete lung expansion, new growths and diaphragmatic hernia.

The collapsed areas are dark red to purple in colour, airless, solid and sunk below the level of the surrounding lung, which is emphysematous, sometimes to a marked extent. They are most common in the lower lobes and posterior portions or the upper ones. Pleural conditions and fibroid or pneumonic changes in the collapsed lung interfere with re-expansion.

The symptoms and physical signs are much the same as in the congenital type (p. 117) and vary with the degree. Usually they come on gradually. Dyspnoea, recession and cyanosis may be very marked. The patches, if large enough, cause impairment of resonance, deficient air entry and diminished vocal resonance. The diagnosis from pneumonic consolidation is generally based on the development of dulness within 24 hours, the air from the obstructed lung being slowly absorbed by the blood, and without further rise of temperature. Crepitations are absent. Small patches give rise to no physical signs for the dulness is obscured by the surrounding emphysema.

The prognosis is that of the disease. In bronchitis of the larger tubes it generally is recovered from. Pneumonia is a common sequel. Avoid prolonged decubitus in the affections liable to cause collapse and encourage deep breathing.

*Hypostatic congestion* is of the same causation and produces the same effects as in adults.

**The Trachea** is rarely malformed, sometimes the œsophagus opens into it. Partial stenosis has occurred. Tracheal obstruction is due to foreign bodies; inflammation of the mucosa, or "tracheitis," commonly part of laryngitis or bronchitis, set up by similar causes and treated in the same manner; or by pressure from without by an enlarged thyroid, a large thymus, intra-thoracic glands which may be caseous, abscess, cyst, dermoid or new growth.

Stenosis causes difficulty in breathing, especially on inspiration, epigastric and episternal recession, a brassy or metallic cough, perhaps inspiratory stridor, feeble breath sounds over the lungs, and sometimes severe cyanosis.



Occasionally the dyspnœa is relieved by leaning forward in the sitting posture. Attacks of paroxysmal dyspnœa are set up by the difficulty in getting rid of viscid mucus, and, rarely, by reflex irritation of the recurrent laryngeal nerve. It may be relieved by belladonna. The voice is clear. If the stenosis is caused by pressure, the usual signs of pressure on the vena cava superior may be also present, and there may be evidence of an intra-thoracic tumour (p. 396). In stenosis of a bronchus the pulmonary signs are unilateral.

**Foreign Bodies in the Air Passages.**—The habit children have of putting objects into the mouth is a potent source of their entrance into the larynx, trachea or bronchi. The actual cause is a sudden inspiration, such as is produced in older children by violent laughter or an unexpected slap on the back. In infants allowed to crawl about the floor the history of the accident is often unobtainable and the mother is positive it cannot have happened. The presence of a foreign body may not be suspected until it is coughed up or found after death. The variety of objects is innumerable.

The effects are immediate and remote. They vary with the size and nature of the object and its location. A roundworm has caused death. Smooth hard bodies, such as beads and buttons, produce the least immediate effect, but may cause recurrent attacks of glottic spasm through being coughed up into the larynx. Soft bodies, and those which are softened by soaking in warm mucus, are less likely to be coughed up. On swelling they become firmly impacted and cause complete obstruction. Pins and other sharp bodies are very dangerous and difficult to extract.

A foreign body entering the larynx causes symptoms of spasm, often alarming. Dyspnœa is great and the child may become black in the face and die from suffocation. The initial choking fit may be absent if the body comes to rest at once. Occasionally, with a small smooth body, there is merely an attack of spasmodic, convulsive or suffocative cough, over as soon as the object passes into the trachea. The symptoms recur if the body is again coughed up into the larynx. A small hard body in the larynx causes paroxysmal cough and perhaps hoarseness, dyspnœa and stridor. If subglottic or in the vestibular region, the voice may be unaffected. Paralysis of the laryngeal adductors has been noted as an immediate effect. In the trachea the body gives rise to respiratory obstruction, recession, stridor and occasionally pain on swallowing. It remains at the bifurcation or passes into a bronchus, more commonly the right as it is larger. In the bronchus it gives rise to unilateral physical signs. The movement of the chest is deficient and there is unilateral recession, resonance is impaired, the breath sounds are weak or absent over the lower lobe or the whole lung, and the voice sounds weak. Less often there is stridulous wheezing on deep inspiration, paroxysmal cough, and pain in the side or epigastrium. Moist sounds develop later. Cough varies and may be absent until septic pneumonia results from decomposition of retained secretions, local ulceration



and microbial infection. The lung mischief terminates in multiple abscesses, some times subpleural, a variable degree of fibrosis, bronchiectasis, empyema and rarely gangrene. Compensatory emphysema and fatal hæmoptysis may occur.

At first the pulse is unduly frequent, the respiration hurried, and the temperature rises about two degrees, a rise which is difficult to explain. Cough and expectoration come on within a week, and a considerable amount of pus may be found in 5-6 days. The later symptoms are those of sepsis:—hectic fever, troublesome cough, purulent and offensive sputa, diarrhœa and malnutrition. On account of the absence of early symptoms the patient may not come under notice until local ulceration and suppuration have begun.

The *diagnosis* is very difficult or very easy. In every case of unilateral lung disease with purulent expectoration and signs of a cavity, a foreign body must be suspected. Emphasis is lent to this probability by a past history of a choking attack, even though remote in time, and the absence of signs of empyema or tuberculosis. Recurrent attacks of choking and dyspnœa afford strong confirmation, if laryngeal mischief can be excluded. An X-ray examination affords conclusive proof in many cases. The lung signs suggest bronchiectasis or tuberculosis.

*Prognosis.*—Untreated patients may get well in time by expulsion of the object or its encapsulation, but quite half of them die from secondary septic inflammation, sudden cardiac failure or some complication. Bronchiectasis is a common sequel. Foreign bodies have been retained for many years. Paroxysmal dyspnœa and fatal glottic spasm may occur at any time.

*Treatment.*—Emetics, patting the back and inversion of the body are dangerous if the body has passed beyond the larynx. They may cause it to pass back into the larynx and bring on fatal spasm. It is a justifiable method if everything is at hand for an immediate tracheotomy and if the object is small and smooth. The proper treatment is by bronchoscopy, using Killian's method of direct laryngoscopy and forceps, if the body is in the larynx. Immediate tracheotomy is safer for bodies lower down, for it stops the risk of glottic spasm and provides a better means of escape for the foreign body, which will often be at once coughed up through the widely opened wound, if the child is inverted. If this fails, an attempt must be made to locate it with a probe and remove it by crocodile forceps, wire loop or other suitable appliance. The tube can be taken out next day, unless portions of a soft and friable body are left behind. It is rarely advisable to attempt removal through the pleural cavity and posterior mediastinum, or through the anterior mediastinum if the object is in the right bronchus. The bronchoscope and the X-rays have added enormously to the means of successfully locating and removing foreign bodies.

**Gangrene** is rare. It is probably as common in children as in adults; especially in marasmic infants under 3 years of age. It is liable to follow



measles, sometimes in conjunction with cancrum oris, and is most frequent in the broncho-pneumonia and pneumonia of marasmic infants. Occasionally it is secondary to septic infection of the mouth and pharynx, caries of the petrous bone or middle ear disease, septic bronchitis and pyæmic conditions generally, foreign bodies in the bronchus, pulmonary infarction, abscess of the lung, chronic pneumonia and tuberculous broncho-pneumonia.

It is the result of thrombosis and secondary infection with a pyogenic organism. The primary cause is in the blood vessels, bronchi or parenchyma of the lungs. In one variety there is a general moist gangrene, diffuse, involving probably a whole lobe, with a dirty green or brownish colour and a gangrenous odour. The lung tissue may be semi-solid or completely liquefied. More often it is of the nature of a patchy necrosis, the result of local thrombosis or embolism, perhaps septic. This affects several areas, is circumscribed, in wedge-shaped patches like infarcts, and free from gangrenous odour. These patches are liable to break down and form abscesses, filled with broken down lung tissue and offensive foetid pus. In late stages the odour may be gangrenous.

The outlook is best in cases due to a foreign body. An isolated patch may be coughed up and end in cicatrisation. Empyema and pneumothorax are occasional sequels.

It is rarely diagnosed during life for it may give rise to no definite indications before death, especially in pneumonic cases. If mouth affections can be excluded, a foetid or gangrenous odour of the breath, prostration and the typhoid state are the most suspicious signs. Older children may expectorate necrotic lung tissue or even have attacks of hæmoptysis. The treatment is on general principles, with a liberal supply of food and stimulants.

**Abscess of the Lung.**—Multiple minute abscesses are found in pyæmia, septic broncho-pneumonia and the secondary infection of tuberculous broncho-pneumonia. Tuberculous cavities are single or multiple and variable in size. A single abscess in the lung is rare; usually the result of necrosis, as in some cases of gangrene, and occasionally due to the breaking down of a mediastinal gland involving the lung. A large single abscess might give rise to signs like those of empyema. The small multiple ones are diagnosed after death.

**Intra-thoracic Tumours** are mediastinal, pulmonary or pleural in origin, arising in the glands, bronchi, lungs, etc. Sarcoma of these structures may be primary in the glands, or secondary in the glands or lung. Lymphadenoma may be primary in the glands or secondary to Hodgkin's disease. Primary sarcoma of the lung is extremely rare. A boy in his fourth year had a round-celled sarcoma of the glands on the right side, softened hæmorrhagic areas, and no secondary growths. He died seven weeks after the onset of symptoms. Dermoids, hydatids, cysts and actinomycosis are great rarities. Dermoids are often latent until puberty. They arise in the



anterior mediastinum and give rise to the signs of a tumour under the upper half of the sternum. Many are discharged through a bronchus.

The *symptoms* are those of pressure on adjacent structures. In the early stages there may be pain in the side or abdomen, cough, emaciation, increasing dyspnœa and unduly frequent pulse. If the diaphragm is involved, there may be much pain and dyspnœa or pleurisy at the onset. In the case mentioned the pain occurred in spasmodic attacks, and was referred to the navel and the right side. The attacks were associated with dyspnœa and cyanosis, and suggestive of diaphragmatic spasm. In later stages the chest bulges and the heart is displaced. Pressure on the vena cava superior causes puffy swelling of the head, neck, chest wall and arms, lividity, slight exophthalmos, swelling of the thyroid, and dilatation of the superficial epigastric and mammary veins. Pressure on the bronchus causes deficient air entry, impaired resonance and subsequent dulness; later, bronchiectasis through infection of stagnant secretions, and then perhaps bronchial breathing.

Because of the pain and physical signs a diagnosis of pleurisy or pleural effusion is likely to be made. Examination shows dulness which may extend to or beyond the sternum, weak breath sounds, diminished vocal vibrations and vocal resonance, and perhaps areas of bronchial breathing over the bronchus in front and behind. In my case the apex of the heart was an inch outside the nipple line, and the liver so depressed that its convex upper surface was felt in the right hypochondrium and the epigastrium and its edge as low as the umbilicus. There was no bronchial breathing and breath sounds were absent over the lower two-thirds of the right chest. A moderate degree of fever has been found in certain cases, and the evidence in favour of empyema has been so strong that portions of rib have been resected. Exploration may reveal the presence of fluid, serous or bloody. In the case referred to gentle aspiration withdrew 24 oz. and 12 oz. of dark, fluid, altered blood on successive occasions. It flowed out quite easily, was a little brighter at the end than at the beginning of the operation, and did not cause faintness. It did not clot and no sarcoma cells could be found in it. Its removal had no effect on the physical signs over the lungs or the position of the heart, and very little on that of the liver. After death it was found to have come from a hæmorrhagic cavity in the growth and not from the pleural cavity.

*Diagnosis* is impossible in early stages. If there is fluid complicating the case, its removal will not definitely exclude the possibility of a new growth as the cause of the effusion. Much stress can be laid on the absence of fluid on exploration, and on the absence of much improvement in the physical signs if fluid is present and is removed. Bloody effusions are often of tuberculous origin. Lymphadenoma may be associated with pyrexia.



Chronic pneumonia, bronchiectasis, fibroid phthisis and tuberculosis must be excluded.

The *progress* is rapid. As the tumour grows it involves more and more lung tissue or compresses it, producing greater dyspnoea and an increase in the physical signs. Death results from interference with the action of the heart, syncope during diaphragmatic spasm, or asthenia. Anorexia, weakness and emaciation are progressive; sometimes there are cough and hæmoptysis. Careful search must be made for a primary focus before accepting a tumour of the lung as primary. The glands may be involved or entirely unaffected, or the tumour purely glandular, displacing and compressing the lung in the process of growth and not infiltrating it. The tumour may start in either the root or substance of the lung. The growth may become adherent to the chest wall and diaphragm, and may involve a rib by direct extension, the heart and pericardium, and adjacent structures.

**Emphysema** is never primary. It is found in the lungs and subcutaneously as a complication of other diseases. General emphysema is due to interference with cough and expiration. It is present in whooping cough, bronchitis, broncho-pneumonia, asthma and laryngeal obstruction. Delicate children develop it by blowing trumpets and wind instruments. In the newborn it may be due to mechanical inflation of the lungs. Pertussis and asthma are the most powerful causes. Compensatory emphysema is a sequel of imperfect expansion of lung damaged or impeded in its action, as by the adhesions of old pleurisy, the contraction of empyema, rachitic and spinal deformity, pneumonia and tuberculosis. Patches are often found surrounding isolated areas of collapse and consolidation.

It is most marked at the anterior edges of the upper lobes, the bases of the lower lobes and at the root of the lung. Emphysematous lung is lighter and paler than normal. The vesicles are quite small, visible through a magnifying glass, and universally distributed in the general type. If the septa give way, the vesicles vary in size from a pin head to a cherry. In rare instances the affection is interstitial or interlobular, and it may extend to the connective tissue of the body, via the mediastinum.

In a well-marked case of general emphysema the chest is fully expanded, barrel-shaped, with little or no respiratory excursion. The diaphragm moves freely and there may be epigastric pulsation from dilatation of the right ventricle. The face and lips show a bluish pallor, breathing is rapid and shallow, the pulse frequent, and there are cough and general distress. The physical signs are high pitched resonance, obliteration of the cardiac dulness, feeble respiratory murmur, and weak or distant heart sounds. The temperature is normal or subnormal. When part of a general disease, acute emphysema usually ends in complete recovery. The prognosis is that of the disease. If there is no evidence of an exciting cause, the outlook



is bad and the child may die from cardiac failure, ante-mortem clotting in the right auricle or broncho-pneumonia.

The treatment is that of the primary disease and in convalescence must be directed to maintaining nutrition and strengthening the right side of the heart by cod-liver oil, iron, dry and equable climate, clothing, etc. The ordinary precautions against attacks of bronchitis must be rigidly enforced.

*Subcutaneous emphysema* appears above the clavicles and spreads downward over the chest, upward to the face, and perhaps over the whole body. The chief causes are obstruction to the bronchial tubes, tracheotomy and artificial respiration, pertussis, rupture of the lung, air tubes or œsophagus, tuberculosis, the bronchitis of measles and pneumonia. It originates in the interlobular and interstitial emphysema of the lungs, a condition which cannot be diagnosed during life. Sometimes the mediastinal tissues are full of air bubbles. Kirschgessner (1904) has recorded a case in a newborn. He ascribed it to extension, via the mediastinum, from an interstitial emphysema of the lungs produced by intra-uterine expiration during extraction, there being no injury to the clavicles or ribs. The child got well in 10 days. Localised emphysema may surround an opening into the pleural cavity, after operation for empyema. Subcutaneous emphysema is generally fatal if associated with tuberculosis or lobar pneumonia.



## CHAPTER XXXVI.

### ASTHMA.

#### *Hay Fever—Catarrhal Asthma—Spasmodic Asthma.*

**Hay Fever.**—Paroxysmal sneezing is sometimes called paroxysmal coryza, vasomotor coryza, or vasomotor rhinitis. It is essentially the same as hay fever, and due to irritation of the terminal filaments of the nasal nerves, occasionally to irritation by a spur on a turbinal. Patches of hyperæsthesia of the nasal mucosa may be found. Many attacks of hay fever appear to be primarily conjunctival. They begin with itching of each inner canthus and irritation of the nose. Examination reveals slight hyperæmia of the bulbar conjunctiva, a fan-shaped arrangement of vessels analogous to pterygium, with redness and swelling of the eyelids, lachrymation and photophobia. It is quickly followed by sneezing, nasal obstruction and profuse nasal catarrh. The attack lasts for about 3 weeks. It differs from paroxysmal sneezing in its extent, the ocular involvement and duration. Thus there are grades of these affections. Possibly in one instance the nasal mucosa is alone susceptible to the irritant, and in the other the conjunctival mucosa is equally or even more susceptible. The youngest case under my observation, was a girl, 3 years old, who had suffered from paroxysmal sneezing since the end of the first year of life. The smell of horses was a definitely exciting cause. The etiology of the condition is the same as that of true asthma.

A spray of adrenalin solution, 1 in 1000, affords immediate but temporary relief. Local tender or hyperæsthetic spots on the nasal mucosa are painted with silver nitrate solution 2 per cent., or touched with a galvano-cautery. Adenoids, enlarged tonsils and hypertrophied turbinals should be removed and proper respiratory exercises prescribed. Dunbar's antitoxin or serum (pollantin) is instilled in fluid form into the eyes, or as a powder into the nose, for hay fever. A mixture of resorcin gr. 2-4, sodium chloride gr. 4 in water oz. 1, with or without acetic acid m. 2, is a valuable nasal lotion. Resection of the nasal nerve has been suggested for intractable cases. High altitudes and sea voyages are useful, if the attack is due to the pollen of certain grasses.

**Asthma** is catarrhal or spasmodic in type. It occurs in paroxysms, with or without pulmonary catarrh and little or no dyspnoea between the attacks. It is not infrequent in infants though often overlooked or



diagnosed as bronchitis. Out of 225 cases collected by Salter no less than 71 began in the first ten years of life and eleven of these began in the first year, one as early as 14 days. The catarrhal type is much more common in infants than the spasmodic one. The earliest case of typical spasmodic asthma under my own observation occurred at 9 months of age, and I have records of 12 others under one year. Cases, which begin after the age of 4 years, are more often of the spasmodic variety. They usually begin in the seventh or eighth year after repeated attacks of bronchitis.

The family history is one of asthma, hay fever, various neuroses, gout or eczema. It is comparatively infrequent among the poor and not uncommon among the better classes, especially the Jews. It occurs in all countries, but is least common in dry climates. It may coincide or alternate with eczema or urticaria. Sometimes it is associated with enlarged tonsils, and cured by their removal, or follows whooping cough.

Instability of the respiratory centre must be regarded as the predisposing cause. The exciting causes may be central or reflex. The chief one is a catarrhal condition of the respiratory system, or a hypersensitiveness. Once an attack has occurred, other paroxysms are induced by reflex causes, such as reflex stimulation of the vagus by aerial emanations, an east wind, the pollen of plants, various dusts and smells. None of these causes can produce an attack unless the predisposition is present. Digestive disturbance, dilatation of the stomach or colon and constipation are so frequent that a special gastric type has been named by Hensch "asthma dyspepticum." Other reflex causes are affections of the nose and nasopharynx, adenoids, foreign bodies in various orifices, teething, dilated heart, and possibly enlarged bronchial glands. Perhaps they are merely coincident. Sometimes a hyperæsthetic area or an unhealthy state of the nasal mucosa, especially œdema of a small area of the upper part of the triangular cartilage opposite and immediately above the level of the anterior third of the middle turbinal bone, has been blamed. Cauterisation of this area has relieved or cured some cases, even when it has appeared perfectly healthy.

There is very little evidence of central stimulation as an exciting cause. Attacks are occasionally produced by emotion and excitement. Toxic poisons, e.g., carbonic acid and those of uræmia, are known to induce dyspnoea and act directly on the respiratory centre.

*Pathology.*—It has been regarded as a spasmodic neurosis, analogous to epilepsy and migraine, or as a lung disease with a marked neurotic element, or due to a specific irritation in the lung which excites bronchial spasm. The actual paroxysm is due to spasm of the muscle in the small bronchioles, or the swelling of the mucosa, or a combination of these two factors. In favour of spasm is the relief obtained by inhalations of chloroform and amyl nitrite, by morphia subcutaneously, and by the ingestion of nitrites which are supposed to act by reducing the contractility of



unstriated muscle. There is sometimes evidence of swelling of the mucosa in the associated swelling of the nasal and tracheal mucosa, and the frequency of bronchitis as an exciting cause. This swelling of the mucosa is due to hyperæmia, vaso-dilatation or angioneurotic œdema. It has been suggested that the œdema is an urticaria of the vagus nerve. According to Dixon and Brodie the pulmonary vessels are devoid of vasomotor nerves. On the whole the evidence is in favour of vaso-dilatation. An attack may be induced by exposure of the chest to cold, or be exaggerated by the inhalation of hot air. It is associated with vaso-constriction elsewhere causing cold skin and extremities, pallor and a feeble small pulse. It is relieved by remedies which induce general vaso-dilatation or local vaso-constriction, by cardiac depressants, and by local or general bleeding. Probably some attacks are due to spasm only and others, in young infants especially, to muscular spasm secondary to swelling of the mucosa.

*Symptoms.*—*Catarrhal asthma* differs from the spasmodic type in the amount of bronchial secretion. It develops during or after an attack of nasal, bronchial or pulmonary catarrh, broncho-pneumonia, pertussis and measles. It is most frequent in the rachitic, the abnormally fat babies, and those with adenoids or enlarged tonsils. The catarrh may be present for a few hours or days before the attack. Paroxysms vary in frequency and usually come on at night. The child is put to bed apparently well, or perhaps a little wheezy and coughing, and in a few hours develops extreme dyspnœa, with anxious face, congested eyes, sweating forehead, pallor, cold extremities and restlessness. On examination there is found marked recession of the soft parts and lower ribs, absence of hyper-resonance, and presence of sibilus and rhonchus, with fine rales at the bases. There may be complete apyrexia, or a rise of temperature 2° or 3° F. The attacks last for some hours or days and gradually subside. After 2 or 3 days the child is well, except for a few rales in the chest and a little cough. Many attacks are quite mild.

A paroxysm may come on during the course of bronchitis which has persisted for weeks or months. It is indicated by an increase in the dyspnœa, a respiration rate of 40-90, a feeble pulse, dry rales, and a little fever. The dyspnœa varies in intensity, is worse at night and in damp weather, and is increased by gastric disturbance. Occasionally it is almost of a chronic type. It may even develop if the temperature falls. The characteristic feature is that the amount of dyspnœa is out of all proportion to the state of the pulmonary signs. The symptoms may last for weeks.

*Spasmodic asthma* is a periodic urgent dyspnœa, more or less sudden in onset, which usually comes on during the night at about 12-3 a.m., and sometimes in the day during sleep. It is preceded by no catarrhal symptoms and lasts for 1-6 hours, occasionally for days, without any rise of temperature. It is ushered in with a feeling of tightness or oppression of the chest, air-hunger and a sense of suffocation. The inspiratory efforts are



violent and expiration is much prolonged, cough is short and dry, and the attack may cease abruptly, sometimes with a fit of coughing. Infantile asthma is often diurnal, occurring at the same hour on successive days. Sometimes the paroxysms are typical in duration and severity, whereas at others they are characterised by periodical attacks of musical sonorous rales, without dyspnœa, for a couple of days or so at a time. During the attack the face is pale, anxious, and becomes more or less cyanotic. The chest is hyper-resonant, in a position of full inspiration with contracted inspiratory muscles, flaccid expiratory muscles and depressed diaphragm. Recession is slight or absent. Breathing is slow and laboured; inspiration is shallow and followed by a pause; expiration is prolonged. The cough is short and dry. As the attack gets worse, the head is thrown back, shoulders raised, hands clenched, eyes suffused and staring, mouth open, lips livid, skin sweating and extremities cold. The child is restless or rigid in a position of orthopnœa. The pulse is small, frequent, feeble and often irregular. On listening to the chest loud inspiratory and expiratory wheezing, sibilus and rhonchus are heard, but no vesicular murmur. Prolonged cases have emphysematous lungs and possibly some dilatation of the right side of the heart. The dyspnœa is less paroxysmal, less severe and of longer duration than in adults. Minor paroxysms are not uncommon. The attacks subside slowly, or may terminate in a fit of vomiting. Pearly white pellets are coughed up.

*Diagnosis.*—Asthma is liable to be mistaken for sudden acute bronchitis, but in this the dyspnœa varies in severity whereas in asthma it maintains the same degree of intensity for some time. The diagnostic features are the more or less sudden onset, the presence of dyspnœa out of all proportion to the physical signs, the absence of fever, the recurrent paroxysms, the almost sudden recovery when apparently likely to prove fatal, and an appropriate family history. Care must be taken to exclude cardiac and renal dyspnœa, and that due to diabetes; the pressure of a retro-œsophageal abscess, enlarged thymus or bronchial glands; dyspnœa from laryngeal affections and foreign bodies in the air passages. In laryngeal obstruction the dyspnœa is inspiratory and in spasmodic asthma it is expiratory. In catarrhal asthma it is more rapid, less laboured, and less expiratory. The spasmodic type is differentiated by the absence of catarrh, recession and fever; hyper-resonance; pronounced expiratory dyspnœa, with slow and laboured breathing; freedom from catarrh between the attacks, and emphysema. Hysterical dyspnœa may be paroxysmal but is not accompanied by distress.

*Prognosis.*—Attacks are rarely fatal but may prove so in infants. Death results from suffocation, cyanosis and prostration. Much emphysema is found after death. Recurrent attacks are more probable in the spasmodic than in the catarrhal type or in those cases in which a definite exciting cause can be found.



The prospects of recurrence and ultimate cure depend on the family history, the social position, the liability to catarrh, and the presence of a definite exciting cause. Most children recover, the attacks subsiding at or before puberty. Cases due to special odours or foods are usually persistent. Recurrence is often periodic. Unfavourable signs are prolonged and frequent attacks, with incomplete recovery between the paroxysms, emphysema and dilatation of the right ventricle. The greater the freedom from respiratory catarrh between the attacks, the better is the outlook, for the less is the damage done to the lungs.

*Treatment.*—Keep the child in as good health as possible, devoting attention to rendering it insusceptible to dyspepsia and catarrhal affections, and to increasing the stability of the respiratory centre. The diet should be simple, nutritious, and consist of small regular meals, avoiding meat extracts and excess of carbohydrates, and not allowing much meat or vegetables. Fruit can usually be given. An anti-diabetic diet has in my experience proved absolutely useless. Exclude foods which definitely cause an attack. Attend to idiosyncrasies in climate, altitudes, town or country. High altitudes are unsuitable for emphysema, but are beneficial in the early spasmodic cases. Choose for these places such as Mont-Dore-les-Bains in France, the Engadine or Colorado. If there is much catarrh, choose a warm dry climate or seaside air and sandy soil, e.g., Bournemouth, Hastings and the Ascot district. Attend to all sources of nasal irritation and local obstruction; remove adenoids and enlarged tonsils. An emetic may cut short an attack, e.g., injec. apomorph. m. 1-5, up to the age of 1 year. Give a purge, if it is due to intestinal irritation.

During the paroxysm of spasmodic asthma keep the child in a warm bed in a room with the windows open. General vaso-dilatation is induced by hot water or vapour bath; hot fomentations or turpentine stupes to the chest; trinitrin or nitrites internally, chloral by mouth or rectum, or alcohol. Constriction of the respiratory mucosa is encouraged by inhalation to cold air from ice in a jug or inhaler, adrenalin 1 in 1000, or the fumes of brown paper, nitre paper, the leaves of stramonium, belladonna or hyoscyamus, or Himrod's powder. Grindelia robusta is occasionally beneficial. Anything which increases the action of the heart increases the dyspnoea, unless the blood pressure also falls. Many cardiac depressants such as tartar emetic, lobelia and tobacco do no good until they produce nausea, faintness or collapse. In catarrhal asthma of infants give inhalations of terebene, pine oil or creosote m. 20 to the pint; and tartar emetic gr.  $\frac{1}{100}$ , trinitrin gr.  $\frac{1}{200}$ , pulv. ipecac. gr.  $\frac{1}{20}$  hourly until relief is obtained. Atropin gr.  $\frac{1}{1000}$  to  $\frac{1}{100}$  subcutaneously is sometimes useful. Morphia stops an attack but should rarely be employed.

Between the attacks give a course of pot. iod. or syr. fer. iod. and cod-liver oil. Arsenic, strychnia and cinchona are useful tonics. Cauterisation of the nose may be tried. The general treatment includes respiratory



exercises, hill climbing, cycling and swimming ; suitable diet and elimination ; Spa treatment and Turkish baths. It is infinitely more important to attend to the general health of the child, to prevent erroneous methods of feeding, to remove local sources of irritation and to cure the catarrhal tendency, than to rely upon measures for relieving or cutting short the paroxysms.



## CHAPTER XXXVII.

### BRONCHITIS AND BRONCHO-PNEUMONIA.

*Acute Bronchitis—Plastic Bronchitis—Chronic Bronchitis—Capillary Bronchitis or Broncho-Pneumonia.*

Catarrhal affections of the bronchial tubes are very common in infants, and, to a much less extent, after infancy. They are dangerous because the young cannot cough and expectorate, and portions of the respiratory area become useless from atelectasis. They are subdivisible into groups according to the degree of extension of inflammation down the respiratory passages. All these affections are infrequent after the third year, except as complications of other diseases.

The main predisposing causes are rickets and impaired vitality. Hence the lack of maternal nursing is a potent factor. The chief exciting causes are chill, rapid changes in temperature, wet and exposure, and cold baths. A few cases are due to direct inhalation of irritants, such as steam from a kettle. Many are secondary to extension of catarrh from the throat, nasopharynx or larynx. They are common complications of measles, whooping cough and influenza, and frequent in typhoid fever, diphtheria and other infectious diseases. Many are due to tuberculosis, or a terminal infection of ileo-colitis and summer diarrhœa. Pathologically they may be ascribed to infection, the bactericidal action of the mucosa being reduced by congestion.

**Acute Bronchitis.**—*Tracheo-bronchitis* is limited to congestion of the mucosa of the trachea and large bronchi, with desquamation of epithelium and exudation of mucus and muco-pus. As the inflammation spreads downward it involves the smaller tubes on both sides, affecting the whole thickness of the walls of the bronchioles and the peribronchial tissues. Pus and mucus can be squeezed out of the smaller tubes after death. It next spreads to the minute tubes and alveoli, causing broncho-pneumonia. In fatal cases of capillary bronchitis there is more or less atelectasis. In the other varieties emphysema is more common. The mediastinal glands are enlarged.

*Symptoms.*—Tracheo-bronchitis and bronchitis of the large and median tubes are comparatively mild affections. They are rarely fatal, except in the very weakly or in the presence of acute infectious disease. Attacks last for about a week or 10 days. General malaise is slight and may be quite absent in mild cases.



In primary cases the onset is more or less acute. In a secondary spread downward the symptoms are usually limited to hoarseness and frequent cough, with substernal pain and perhaps spasmodic. On auscultation there may be no abnormal sounds; a little roughness of the respiratory murmur; or rhonchus and sibilus, especially in the subclavicular and interscapular regions, according to the degree of extension. The breathing is weak and more frequent, wheezing may be felt, and the note is resonant. Older children may complain of a feeling of tightness or constriction across the chest. Respiration is increased in proportion to the fever, mucosal swelling and secretion. The cough varies in severity; at first dry and hard, later loose and moist. Rales may be heard in the chest. Typical bronchitic sputa, occasionally blood-stained at the onset, may be expectorated by older children. Those under 6 years of age generally cough the sputum into the throat and swallow it. Anorexia, dyspepsia, and constipation or diarrhoea, are often present. Vomiting may be induced by coughing or the irritation of sputum in the throat. The temperature runs up to 101-103° F. in generalised cases of medium severity, depending on the nature of the infection rather than the actual pathological changes. It comes down gradually in a few days.

*Treatment.*—Delicate and tuberculous children must be protected from attacks by careful feeding and general hygiene. It is a mistake to coddle infants in hot stuffy nurseries. They should be gradually inured to free exposure to fresh air and all changes in temperature, provided they are properly clad and not exposed to strong cold wind. At night they should sleep in a large airy room with the window open. The bed must be screened from direct draught and a fire lighted in the room, if the weather is cold. The temperature of the room should not exceed 65° F. in the first year, 60° F. in the second, and may be permitted to fall to 50° F. in later years. The child should wear a flannel or Jaeger nightdress, be well covered with clothes and allowed a hot water bottle. Up to the fifth year of life children must be guarded from exposure to measles, whooping cough or influenza, for the bronchial catarrh common in these diseases is liable to end in fatal broncho-pneumonia. Delicate infants must be protected from the risk of catching the ordinary infectious cold. If the child develops catarrh of the upper respiratory passages whether in the nose, naso-pharynx, tonsils, pharynx or larynx, it must be kept in bed as long as there is fever and indoors until practically well. The temperature of the room should be kept at about 65° F. While in bed the position should be often changed. Debilitated infants must not be allowed to lie on their backs all day. They should be nursed at intervals or put in the prone position.

An attack may be cut short by a hot bath, diaphoretics, a dose of calomel or castor oil, and a small dose of Dover's powder. Phenacetin and aspirin are sometimes useful. If the mischief progresses, apply stimulating counter-irritants to the chest and put on a pneumonia jacket of cotton wool.



Poultices are better avoided. If there is much fever, compresses at the room temperature can be applied to the chest every  $\frac{1}{2}$ -2 hours. Emetics and expectorants do more harm than good in the first year of life. They are liable to upset the stomach and depress the child. Vinum ipecac. may be given in drachm doses every  $\frac{1}{4}$ -1 hour until it acts as an emetic on strong healthy children whose tubes are blocked with secretion.

Inhalations of simple or medicated steam relieve the feeling of tightness and induce secretion. Later on turpentine, creosote and the oil of pine or eucalyptus can be given, sprinkled on cardboard or lint, for 10-15 minutes at a time t.d.s. In early stages a mixture of vin. ipecac (or vin. antimon.), pot. citrat. and syr. tolu in small doses is unlikely to do harm. Ammonium chloride and potassium iodide can be given in a day or two to render the secretion more liquid, and later on drugs such as squills, creosote and terebene. Cardiac and respiratory stimulants, as in broncho-pneumonia, may be needed.

As a general principle mild cases require little treatment except careful feeding and nursing, and more severe ones are treated on the same lines as broncho-pneumonia. In older children minute doses of opium may be given at the onset and then ammonium chloride, or terebene m. 2-5 on sugar, and inhalations of creosote or turpentine. Phenacetin and Dover's powder are useful if the cough is troublesome and secretion scanty.

**Plastic Bronchitis.**—A fibrinous or membranous secretion is commonly due to extension of diphtheritic membrane from the larynx. Sometimes it is primary, acute or chronic, and of doubtful causation. Such cases may be due to the pneumococcus or to the diphtheria organism. The membrane may be limited or extend widely, even to the minutest tubes. It is partially loose where it is becoming separated, previous to being coughed up. Patches of collapse, broncho-pneumonia and emphysema are often present.

Cases are ushered in with headache, cough, vomiting, and the feeling of a lump in the chest. The aspect is dusky and the sputum blood-stained. The symptoms are those of bronchitis of a severity varying with the degree of extension of the membrane. The chronic form is preceded by several attacks of prolonged bronchitis and is characterised by severe dyspnœa, coughing fits, often paroxysmal and associated with suffocation, and the expectoration of membrane. In prolonged cases anorexia, pallor and wasting may be present. Examination of the chest yields signs of bronchitis and secondary infection. If the mischief is unilateral or unilobar there may be impairment of resonance, large rales, and bronchial breathing. S. West (1908) reported a case of a girl, 11 years of age, who had 7 attacks in 4 years, in which were found absent breath sounds and vocal resonance, and stony dulness due to collapse of the lung, with extreme displacement of the heart and mediastinum. These physical signs disappeared when the cast was coughed up.



The diagnosis depends on the expectoration of the membrane. It is often coughed up in balls and is not recognised as membrane unless floated out in water. That from the trachea and larger bronchi looks somewhat like cooked macaroni and from the smaller tubes like vermicelli. It may form an arborescent mould of the whole bronchial tree or the branches of one lobe. A central cavity is exceptional. The membrane is composed of fibrin, leucocytes and epithelial cells, possibly ciliated ones from the trachea. It is soluble in lime water and alkalies.

Acute cases last a few weeks but are usually fatal. Chronic cases may continue for months or years, with intervals of days or weeks between the attacks. The temperature is generally raised and at its maximum just before the cast is coughed up. Afterwards the child improves and may be apparently well till membrane forms again and gives rise to dyspnoea.

These cases are treated by inhalation of alkalies, creosote, terebene or turpentine; potassium iodide and pilocarpin internally; and residence in a warm dry climate.

**Chronic Bronchitis.**—Chronic bronchitis is uncommon in children. It may be a sequel of an acute or sub-acute attack in a delicate child or one living in bad hygienic surroundings. Predisposing factors are deformity of the chest from rickets and spinal curvature, congenital heart disease, mitral stenosis and regurgitation, tuberculosis and bronchiectasis. A few cases possibly are due to congenital syphilis.

Cough is the main symptom. It is more or less chronic, worse in cold weather and winter, worse in the early morning from accumulation of secretion in the tubes during sleep, and often paroxysmal from bronchial spasm and suggestive of whooping cough. Expectoration is usually scanty. The general health is little or not at all affected. Vomiting may be induced by cough, especially in the morning after breakfast. Bronchitic rales may be present in the morning and during exacerbations. The chest becomes emphysematous.

The diagnosis is based on the history, emphysema, bilateral physical signs, and the absence of signs of tuberculosis such as nocturnal fever, wasting, and tubercle bacilli. Pertussis must be excluded. The duration of the disease is indefinite and there is great liability to attacks of acute or sub-acute bronchitis.

These patients are treated by climate, clothing, food and cod-liver oil. Terebene or hydrate of terpene, m. 2-5 on sugar quartis horis, relieves bronchial spasm, lessens dyspnoea and makes the secretion less and thinner. Pot. iod. and ammon. chlor., creosote and expectorants, and alkalies are also useful; and digitalis in cardiac cases.

**Broncho-Pneumonia.**—*Syn.*: *Capillary Bronchitis*—*Catarrhal or Lobular Pneumonia*—*Acute Infantile Broncho-Pneumonia*—*Disseminated Patchy Pneumonia*—*Broncho-pneumonitis*. Broncho-pneumonia is the best name for this disease for it is a catarrhal inflammation of the respiratory



tract which extends to the minutest bronchioles and to the adjacent alveoli of the lungs. It causes peribronchitis, inflammation of the interstitial tissue of the lungs, frequently of the pleura, as well as patches of pneumonia. The name "catarrhal" suggests the common mode of production and "lobular" distinguishes it from lobar pneumonia, while "capillary bronchitis" indicates that the minute tubes are affected.

The affection is essentially one of infancy and is rare after the fourth year. Quite half the cases occur in the first year of life, one-third in the second year of life, and one-tenth in the third year. During the first year the cases are equally distributed in each quarter. After infancy it is rarely seen except as a complication of other diseases. In addition to being one of the most common, it is one of the most serious diseases of infancy. Its gravity is increased in the very young, the badly nourished, and those weakened by grave illness. Two-thirds of the cases occur in the winter and spring. It is both more frequent and more serious among the poor because of debility and lack of care.

The general etiology is that of bronchitis. About one-third of the cases are primary and the remainder secondary to bronchial catarrh. The susceptibility of infants to this form of lung disease is due to the incomplete development of the alveoli during the early years of life, for they are not fully developed till the third or fourth year. Primary broncho-pneumonia may result from chill, but is most commonly a pneumococcal infection, such as would have induced pneumonia in older children.

*Bacteriology.*—The majority of the primary cases are due to the pneumococcus alone, and the rest to the pneumococcus in conjunction with other organisms, especially the streptococcus. In secondary cases the streptococcus is the most common, alone or with the pneumococcus. The influenza bacillus, diphtheria bacillus and *B. coli* have also been found.

*Classification.*—On grounds of severity there are four groups. The first is fatal in 12-36 hours. The second group lasts for 6-10 days. The third is prolonged for 3 or 4 weeks, occasionally 6 or 8 weeks. The fourth is cachectic in character, with little or no fever and often evidence of little or no disease during life. S. West divides cases into three types :—(1) A secondary broncho-pneumonia of gradual onset with hectic temperature, prolonged course, relapses, and termination by lysis ; (2) Cases of sudden onset but otherwise resembling the first group ; (3) A primary variety with no antecedent affection of the bronchi. This is characterised by sudden onset, short duration, persistently high temperature, no relapses, and termination by crisis. It is usually pneumococcal and clinically identical with lobar pneumonia of adults from which it only differs in extent of physical signs. According to West it is really a patchy, pneumococcal pneumonia. On anatomical grounds we may speak of a "capillary bronchitis," with no physical signs of consolidation during life, though small patches are found after death ; "lobular pneumonia," in which there are



definite small patches of bronchial breathing, possibly aggregated into a sufficiently large mass to cause a local area of dulness; and a "lobar" form, in which the patches are so large and confluent as to closely resemble lobar pneumonia.

Some cases are characterised by intense pulmonary congestion, or by rapid dilatation and failure of the heart, or by high temperature and acute delirium, or by asthenia or toxæmia. Thus we may speak of congestive, cardiac, nervous, asthenic and toxæmic types. On the whole it is advisable to recognise these different clinical groups and that they may occur in primary or secondary varieties, and in any type of anatomical distribution.

*Morbid Anatomy.*—In capillary bronchitis the lung is vesicular throughout and engorged at the onset, and shows patches of collapse. In broncho-pneumonia irregular areas of consolidation are present. The pneumonic condition may be peribronchial, limited to the air cells round the smaller tubes; bronchial and vesicular simultaneously; or in the form of irregular lobular pneumonia with no definite relation to the bronchioles. Isolated lobules or groups of lobules appear as prominent purplish patches on the surface. These patches are more or less grey and give the lung a granular feel, like "frog's spawn," on passing the finger over the cut surface. Exuded fluid can be squeezed out of the bronchi and inflamed lobules. Isolated patches on the surface are surrounded by emphysema. In the lobar type the consolidated area is not as sharply defined as in true pneumonia, and portions of the lobe escape. Thus the different stages are acute congestion, commencing consolidation, patchy red and grey hepatisation, and general grey hepatisation. In the last stage isolated pneumonic patches break down and form small cavities containing pus; the vesicles are full of leucocytes and the alveolar septa much thickened; vesicular and interstitial emphysema are present.

Broncho-pneumonia is generally bilateral, in the posterior parts of the lower lobes, especially in infants under one year kept constantly on the back. Often it has a paravertebral distribution up to the middle of the scapula. It rarely extends beyond the axilla. Both lungs are affected in over half the cases and the base five or six times as often as the rest of the lung. It may be unilateral and affect any part, notably that portion of the left upper lobe in front of the pericardium.

*Symptoms.*—In the primary cases the onset is sudden and acute with a rise of temperature to 103-105° F., severe dyspnœa, great prostration and occasionally convulsions. Vomiting occurs in two-thirds of the cases and there is often pain in the side. The fever shows remissions of 2-3° F. every morning but is sometimes higher in the morning than at night. The course is that of true pneumonia ending by crisis and it is rarely fatal. In the secondary type the onset is often sudden but generally more insidious. Many attacks are ushered in, or preceded for a day or two, by diarrhœa and vomiting. Usually after a variable period of slight bronchial catarrh the



child becomes rapidly and seriously ill with cough, fever and respiratory distress. The temperature rises to about 104° F., and ranges irregularly between that and 102° F. A mild attack lasts 3-5 days and subsides by a fairly rapid lysis. In 10 per cent. the fall of temperature is so rapid as to deserve the name of crisis. Often a pseudo-crisis and secondary rise occur. In asthenic cases there may be little fever and even a subnormal temperature throughout, although lung consolidation is extensive. Relapses are apt to occur in the secondary variety and may be similar in character and course to that of the primary type. Remissions and exacerbations are common, and fever may continue for several weeks and subside gradually.

*An Acute Congestion of the Lungs* sometimes occurs in young infants. The onset is sudden with high temperature, dyspnoea, shallow breathing, cyanosis and prostration. The symptoms indicate severe toxæmia and no suggestive lung signs may be found. The babe is listless and apathetic. The fever and symptoms may abate in 2 or 3 days, or clear up entirely within a week.

The general symptoms of broncho-pneumonia are very variable. In a well-marked case the facial aspect is anxious, of a livid pallor or cyanosed, with injected eyes. The nails are bluish and the skin a dull leaden hue in bad cases. The child is restless and the cry short and suppressed. Respiration is hurried and laborious, 60-90 per minute, or more when the small bronchi are invaded. The early alteration in the ratio of respiration to pulse rate is a valuable sign. The alae nasi dilate and the respiratory obstruction causes exaggerated movements of the chest, with considerable recession of the soft parts and especially of the lower ribs in the lateral regions. The child is greatly distressed by the difficulty in breathing. This distress is an important feature for it is absent in pneumonia, although respirations may be as frequent, but in pneumonia there is not the same obstruction in the air passages. The pulse rate ranges from 140-200. It is decreased in volume and often irregular. The tongue is furred, appetite lost, abdomen often distended from tympanites, and diarrhoea or constipation present. The child is irritable, listless and drowsy, or languidly restless. Prostration increases with toxæmia and greater involvement of the lung, and may pass into the typhoid state.

In the very malignant type, most frequent in young infants, the mechanical congestion is general, obstruction complete, and toxæmia marked. The temperature is over 104° F., breathing hurried, pulse rapid and weak, prostration grave, and cough often absent. There may be no physical signs; sometimes vesicular breath sounds are absent or exaggerated. Death may result from asphyxia or from toxæmia. In the latter case there is much collapse and a temperature of only 100-101° F., or dulness and apathy pass on into stupor and convulsions. These cases may be spoken of as acute congestion of the lung, acute capillary bronchitis, or possibly *la maladie de Woillez* (p. 420).



In the next most serious type the child is more robust or the fever, oppression, asphyxia and signs of toxæmia are less marked and of more gradual development. It may be a sequel of the previous variety. It may clear up in about a week, pass on into the next variety or increase in severity with constant cough, hyperpyrexia, and death in a few days from asphyxia, respiratory or cardiac failure. In the more favourable cases the onset is less abrupt and the physical signs those of bronchitis until the mischief extends downward. Then the temperature rises to 102-104° F., the pulse to 140-160, respirations to 60-90, and the child is restless and more ill. Cough is at first less hard, but then becomes incessant, hard, painful, and perhaps paroxysmal. Mucus is coughed up into the pharynx and swallowed, or only into the larynx and is then sucked back into the lung; it often causes vomiting. The alae nasi dilate, the face is pale and the lips blue, and the inspiratory muscles are strongly exerted. The expiratory grunting of pneumonia is heard. On the third to the fifth day the symptoms subside to a great extent. Pulse rate, respirations and fever are less. The paroxysms of cough and restlessness are less severe and everything seems favourable to ultimate recovery. The prognosis may be satisfactory, but frequently exacerbation ensues in one or more days, from the development of a fresh patch of consolidation. And so the disease may run on for weeks at a time, the inflammation subsiding in one place and breaking out in another a few days later.

The breathing is most frequent in capillary bronchitis. Its rhythm is altered in all varieties, it is often typically Cheyne-Stokes. As the child gets worse it becomes more frequent, shallow and irregular, all the accessory muscles are brought into action, recession is great and cyanosis extreme. The eyes are glazed and half-closed, the head often retracted, the pulse small and running, and the extremities cold and livid. The facial aspect exhibits ominous pallor, cold sweats, and moaning apathy. Cough and crying cease, meteorism is often troublesome, mucus collects in the throat and death results from convulsions or asthenia. The temperature is often low and breathing very feeble towards the end.

Cyanosis is a grave and important symptom and often associated with dilatation of the right side of the heart. Convulsions are uncommon at the onset but apt to occur and prove fatal in whooping cough cases. Inability to recognise parents, excitement and delirium occur in bad cases. Occasionally symptoms such as head retraction, drowsiness, torpor and vomiting, suggest meningitis. Intestinal catarrh or ileo-colitis is often troublesome and intractable, the stools containing mucus and undigested food. It is due to overfeeding, impaired digestion, fever and toxæmia. The urine is febrile in character, contains albumin in high fever, and sometimes acetone.

The *physical signs* of capillary bronchitis are limited to weak breathing and fine rales scattered all over the lungs; perhaps more marked



in some parts than in others and associated with impaired resonance. Often only bronchitic signs are present though multiple minute areas of consolidation are found after death. Patchy consolidation is indicated by the tympanitic note, local aggregation of sharp rales, and harsh breath sounds, usually over the lower lobe and near the spine. Bronchial breathing and bronchophony afford definite confirmation. Bronchophony is more easily heard than bronchial breathing. The heart sounds may be unduly well conducted to the back. Dulness is not a reliable or constant sign; it may be absent or obscured by surrounding emphysema. The cardiac dulness may be diminished by emphysema of the anterior borders of the lungs. Extensive consolidation gives rise to the same signs as lobar pneumonia, but the dulness is less definitely circumscribed and often not limited to one lobe. It is less marked because more superficial, and less complete because of intervening areas of healthy or emphysematous lung. Usually crepitations are present throughout for air enters more or less into the bronchioles and the air cells, except in collapsed lobules, whereas in true pneumonia the cells are apt to be completely bunged up with exudation. Bronchial catarrh is found in the rest of the lung. In protracted cases the breathing may become cavernous and the signs of consolidation are more complete. It is often necessary to make the child cry during auscultation, in order to get it to breathe deeply and for the sake of estimating the voice resonance. Physical signs are generally present in a week.

*Diagnosis.*—Primary broncho-pneumonia closely resembles pneumonia in its onset and course. Usually it is of shorter duration, if limited and localised. Frequently there is more cyanosis and dyspnoea, more troublesome cough, more laboured breathing, greater remissions of temperature, and disseminated lesions. Sometimes differential diagnosis is impossible. Acute malignant capillary bronchitis may be mistaken for cerebrospinal meningitis or a specific fever with suppressed eruption. Symptoms are often more reliable than physical signs, and the diagnosis has to be based on the rapid breathing, high temperature, prostration, cough and cyanosis. Examination of the chest may prove useless, unless secondary pneumonia has developed. The disease is often unrecognised in the first few months of life, for it may occur with little cough, dyspnoea, cyanosis or fever. Localisation is a very important sign. Limitation of catarrh to one lobe or lung indicates broncho-pneumonia or tuberculosis. In bronchitis the patient is less languid and ill, cough more forcible and paroxysmal, rales coarse and dulness absent. Friction and rales are much alike in many cases and both may be present. Pain is a sign of pleurisy. The signs of effusion must be looked for, if the symptoms do not abate in a week or two and the dulness persists or increases. In the first three months of life atelectasis is often associated with broncho-pneumonia, but atelectasis alone is rare after this age. Some of the apyrexial cases in the first year of life simulate asthma but the latter disease is rare and, though the breathing is rapid, there is little distress. Tuberculous broncho-pneumonia cannot be



distinguished by either symptoms or physical signs. Many cases run an acute course and are only recognised after death. It should not be diagnosed hurriedly for simple broncho-pneumonia may run a prolonged course of 3 months and yet end in recovery. In many instances unsuspected miliary tuberculosis is found in fatal cases of supposed capillary bronchitis.

*Prognosis.*—The mortality of hospital cases is very high, from 30-75 per cent. according to the severity of the cases admitted. Compared with pneumonia it is a most deadly disease. On the other hand the mortality in private practice ranges from 10-30 per cent., according to the social class of the patient. The younger the child the worse is the prognosis, and the mortality is increased by rickets, deformity of the chest, debility, diarrhoea and vomiting. It is bad in diphtheria and scarlet fever, less dangerous in measles unless it occurs before the rash, less in pertussis, and good after simple bronchitis. Primary cases are not so fatal as secondary ones. Streptococcal infections are worse than pneumococcal ones and are usually associated with a more hectic type of temperature, which is consequently rather a bad sign. The percentage mortality increases with the rise of temperature above 104° F. The disease is more serious if the pulse rate is over 180, and if the ratio of respiration to pulse is 1-2, or 2-3. It increases with the amount of lung involved, but the less definite the area of consolidation the worse is the prognosis, for the disease is less fatal in the lobar pneumonic type. Late convulsions are almost invariably of fatal significance or may be the terminal event. Death is usually due to asthenia, asphyxia, and sudden or gradual failure of the heart. A few die from hyperpyrexia or meningitis, and some from secondary empyema. The chief bad signs are increasing restlessness, distress, cyanosis and dilatation of the right side of the heart; the child ceasing to shed tears, becoming drowsy and lethargic; the pulse faster and weaker; increasing dyspnoea and recession; tympanites, vomiting and diarrhoea. A suppressed and inefficient cough and an unusually low temperature are signs of debility and deficient reflex sensibility. As long as food is taken and digested well, the child is likely to recover. Four-fifths of the cases recover or die within 3 weeks and after 4 weeks the outlook is bad. Sometimes the disease may continue for 6-12 weeks with continued fever, or irregular pyrexia and exacerbations; wasting, anæmia and debility; anorexia, vomiting and ill-digested stools; dry inelastic skin, bed sores and punctate hæmorrhages. Such cases simulate phthisis. The general state is of more value than the physical signs as regards the immediate prognosis; but the duration of the illness is greater, the greater the extent of the consolidation. Death may occur within 12 hours. A child of 8 months became blue and short of breath in the morning, had convulsions at mid-day, was collapsed at 4 p.m. and died at 6 p.m. Relapses and recurrent attacks may produce chronic interstitial pneumonia. These children are liable to recurrent attacks.



*Complications and Sequelæ.*—Vomiting and diarrhœa are common, especially in the summer, and very fatal in infancy. Collapse of the lung is frequent in the weakly and almost invariably present in fatal cases. It produces increased dyspnœa, sometimes sudden and fatal, increasing cyanosis, and dulness. Periods of great depression, sudden in onset and disappearance, may possibly be due to this cause. Fibrinous pleurisy and serous or purulent effusions, often bilateral, are not uncommon. Emphysema is constant; minute abscesses, due to necrosis of pneumonic patches, frequent; and gangrene occasional. Other complications are otitis media, purulent meningitis, peritonitis, purulent arthritis, acute periostitis, and pyæmia. It is not always easy to be certain which is the primary disease. Otitis media may give rise to the usual symptoms or only be recognised by the appearance of otorrhœa.

In all varieties of bronchitis and broncho-pneumonia the mediastinal glands are engorged and rendered susceptible to tuberculous infection. Many cases of tuberculosis can be traced to an attack of this kind. General tuberculosis is a rare complication, but both it and chronic tuberculosis are not uncommon sequels. It is difficult to distinguish chronic broncho-pneumonia of an upper lobe from phthisis. Recovery may be prolonged on account of anæmia, severe dyspepsia, or dilatation of the heart. For some months these children are liable to catch cold and get recurrent attacks of bronchitis, finally developing chronic bronchitis and emphysema.

*Treatment.*—The prophylaxis is that of bronchitis generally. During an attack the child must be kept in a well ventilated room at 60-65° F., or 5° F. higher if very weak. Open air treatment is very beneficial in secondary cases, when the attack is not due to exposure to cold. By this is meant a free supply of pure air and not reckless exposure to damp and fog. The good effects are produced very quickly. The child becomes less restless and irritable, the face less flushed, the lips less blue; the pulse and respiration rates fall; the appetite improves, the child sleeps better and the strength is well maintained. It is contra-indicated by convulsions and laryngitis.

The diet must be liberal, nutritious, easily digested, and given in small quantities. It is of the utmost importance to guard against tympanites. In the first year of life breast milk is the best. If that is not available give diluted or peptonised milk, sweetened with milk sugar and strengthened with plasmon or other form of dried casein. Whey, albumin water, veal or chicken broth and a liberal supply of water can be given.

Emetics are only justifiable in the case of robust infants with excessive bronchial secretion and respiratory obstruction. At the onset of an acute attack with fever give a bath at 90-95° F., reduced to 80° F. or 75° F., for 5 minutes. First give the child a stimulant, then sit it in the water, supporting the back with the hand, and do not let the water rise above the level of the lower ribs. Sponge it freely, wrap it up in a warm blanket and put it back to bed. A warm bath night and morning can be given if the temperature



is over 102° F. At the onset a poultice of linseed, one part of mustard to eight of linseed, bread or bran, can be used. It must be sufficiently large to cover the back of the chest and not extend on to the front of the chest or abdomen. Apply one every 20 minutes for a couple of hours, but be sure that the temperature is not too high, and then put on a cotton wool jacket. The ordinary measures of counter-irritation are preferable in mild cases.

If seen in the early stage give tr. belladonnæ m. 4, or extract gr.  $\frac{1}{4}$ , every 3 or 4 hours. It reduces the bronchial spasm, diminishes the amount of secretion in the tubes, and is both a cardiac and respiratory stimulant. If it does not produce a definite result in 24 hours it should be discontinued. The belladonna should be combined with diuretics and diaphoretics. If the child is not seen for 12-24 hours after the onset give a mixture of ammon. carb., pot. iod. or ammon. chlor., tr. nuc. vom., syr. tolu. and chloroform water. Under 1 year of age gr.  $\frac{1}{4}$ -1 of the carbonate can be given every 4-6 hours. Later on stimulating expectorants, especially squill and senega, are required. The squill is also a cardiac tonic. Small doses of phenacetin are useful in nervous cases, severe headache and high fever. Keep the bowels open by small doses of calomel or grey powder. Strong purges are apt to set up fatal ileo-colitis.

The bronchitis kettle can be used, if the atmospheric air is very dry and the cough dry and hard. If it is used, the child must be put in a half-tent so as not to keep out too much fresh air. The vapour from the steam kettle is apt to condense and become cold, and the child then inhales a vapour which is more likely to do harm than good. Inhalations are best given by sprinkling turpentine, eucalyptus, etc., on lint or flannel and holding it in front of the child's nose and mouth for 5-10 minutes every 2 hours. If there is much tympanites starve the child entirely, unless very weak, giving nothing but water for a time. Gastric distension is relieved by lavage, and intestinal distension by the passage of a rectal tube or an enema of asafoetida and water. Nux vomica or strychnia can be given in large doses. Severe diarrhoea is treated on dietetic lines, and occasionally it is necessary to take the grave responsibility of giving minute doses of opium to check excessive peristalsis.

High fever is treated by hydrotherapeutic measures (p. 90). For nervous symptoms associated with moderate fever, a hot bath gives more relief than a cold one, but cold is necessary if the temperature is high.

For sudden collapse due to rapid distension of the heart from toxæmia or mechanical obstruction of the lungs, or due to atelectasis, give a mustard pack or bath, and inhalations of oxygen or amyl nitrite, and stimulants freely. Oxygen cylinders must be at hand in every bad case. The most useful stimulants are brandy, strychnia, nitroglycerin, camphor and caffeine. If the collapse is definitely due to cardiac failure, large doses of carbonate of ammonia, sal volatile or ether should be given. Strophanthus and digitalis help to prevent dilatation of the heart but are, I think, dangerous



if it is already present. Dilatation of the right side of the heart, sufficient to cause dyspnœa and cyanosis, is best relieved by applying 1-3 leeches about the level of the right nipple.

For failure of respiration give an injection of atropin gr.  $\frac{1}{1000}$  -  $\frac{1}{500}$ , strychnia gr.  $\frac{1}{400}$  -  $\frac{1}{100}$ , and caffein gr.  $\frac{1}{10}$ . Such cases should be encouraged to breathe deeply by making them cry, and by alternate hot and cold douching of the chest. For passive congestion at the bases of the lungs dry cupping is of great use. An important point in the nursing of these babies is not to allow them to lie on their backs all day long.

In the convalescent stage, all the hygienic precautions suitable for incipient tuberculosis should be adopted. The dilatation of the right side of the heart, especially after whooping cough, requires treatment by tonics and moderate exercise. For chronic cough give terebene or creosote preparations. The ordinary convalescent requires cod-liver oil, iron preparations, arsenic and small doses of quinine. Choose for these patients a warm and dry climate, and a high altitude in hot weather.

Mild cases, especially primary ones, recover under the simplest methods of feeding and nursing. In those with marked mechanical obstruction, pay chief attention to relieving the congestion of the lungs by drugs and counter-irritation to the chest. If the toxæmic symptoms are pronounced, rely upon alcohol, strychnia and elimination.



## CHAPTER XXXVIII.

### PNEUMONIA.

*Syn. : Pneumonic or Pneumococcal Fever—Pneumococcal Infection of the Lungs—Croupous or Fibrinous Pneumonia—Pleuro-Pneumonia.*

Pneumonia is an acute infective disease, characterised by a definite course and type of lung inflammation. It is a general pneumococcal infection for the organisms and toxin are present in the blood. Death is usually due to toxæmia and heart failure. It affects healthy children, is almost always primary, of sudden onset, typical course, ends by crisis, and is lobar and sharply defined in distribution.

Its causation is the same as in adults and its infective character is clearly established. Groups of cases are sometimes seen in hospital practice, showing a certain amount of epidemicity. Two-thirds of the cases occur in the winter and spring months, either because of the changes in temperature or lowered resistance due to indoor life and winter catarrhs.

*Ante-natal pneumonia* has been reported. A child, born 36 hours before the death of the mother from double pneumonia, lived 2 days. Lobar pneumonia was present and diplococci were very numerous (Levy, 1890). Other cases of ante-natal pneumonia may be syphilitic in origin. True *pneumonia in the newborn* has been reported at 8 days of age; the whole of the left lower lobe showing red hepatisation (Townsend, 1889). Pneumonic affections in the newborn are usually of the nature of broncho-pneumonia, due to inspiration of discharges, cold, etc. Pneumonia was regarded as exceptional under 2 years of age and Parrot went so far as to deny its existence. Now it is recognised to be quite frequent and even more common at this period than later in life. Out of 147 cases, 45 were under 2 years (Dunlop, 1908). Other observers have found it almost as frequent. Riviere collected 154 cases under 10 years of age: 20 in the first, 46 in the second, 38 in the third, 27 in the fourth, 18 in the fifth, and 5 in the ninth year. These figures suggest that cases of primary broncho-pneumonia may have been included. They are not confirmed by post-mortem evidence and diagnosis may be erroneous. Pneumonic affections in infancy were formerly regarded as due to broncho-pneumonia; now there is an undue tendency to regard broncho-pneumonia as pneumonia. The sex incidence is 3 boys to 2 girls.



*Bacteriology.*—The pneumococcus is a normal inhabitant of the nasopharynx and adjacent parts, and is found in the saliva and lungs of healthy children. It sets up disease when the vital resistance is lowered, and is found in the blood in pathological states. In certain conditions it is highly resistant and it lives for a long time in sputum. It is easily killed by chemical agents and moist heat. As an infective agent it has a short life history, and the rapid development of an antitoxin leads to a short acute illness and well marked crisis. It forms little or no soluble toxin. It may produce a general pyæmic state giving rise to broncho-pneumonia, empyema, arthritis, peritonitis, pericarditis, infective endocarditis, abscess of the lung, thrombosis of the lateral sinus, meningitis, otitis media, etc. Davies and Langdon Brown collected 39 such cases, of which 21 were under 12 years of age and none of these due to pneumonia. The presence of the organism in the blood is of grave prognostic significance.

*Morbid Anatomy.*—The anatomical changes are the same as in adults, but the line of demarcation from the unaffected tissue is less well defined and the cut section looks less granular. No intervening unaffected lobules are found as in broncho-pneumonia, but broncho-pneumonia may occur round pneumonic patches or may affect the opposite lung, especially under 2 years of age. In other words the pneumococcus may produce both broncho-pneumonia and true pneumonia at this age and even simultaneously. Pleurisy of all grades is associated with it. In pleuro-pneumonia the affected lung is covered with a thick layer of lymph. The left lower lobe is involved in about one-third of the cases, and the right lower lobe in about half that number. The right upper lobe is as susceptible as the right lower lobe, and the left upper lobe much less often involved. The disease may involve the whole lung, both apices or portions of both lungs, or be limited to one lobe, part of a lobe or a single central area. The proportion of cases on the right side to those on the left is 9-8.

*Symptoms.*—An acute idiopathic congestion of the lungs, probably a degree of acute pneumonia or acute capillary bronchitis (p. 412), is known as *la maladie de Woillez*. Pneumococci alone, or associated with staphylococci or streptococci, were found in 9 out of 14 cases by Carrière, and the pneumococcus was found in the blood in 7 out of 10. Leucocytosis was present. The attack is ushered in by chilliness or rigor, pain in the side, rapid and shallow breathing, a moderate degree of fever, and cough. The physical signs are impaired resonance, feeble and sometimes bronchial breath sounds, and fine crepitations.

The clinical picture of pneumonia in the young differs from that in adults because of the impressibility of the nervous tissues. After the seventh year it more closely resembles the adult type. In infancy the symptoms may be very alarming and the cause obscure. Occasionally cough is slight and respiration is little accelerated. The constitutional



symptoms depend on the toxæmia and often bear no proportion to the severity or extent of the local inflammation.

The onset is sudden with vomiting, malaise, pallor, and shivering or a feeling of cold. Vomiting occurs in two-thirds of the cases. It may be single or repeated for one or two days and may be very violent. Occasionally convulsions or a rigor occur; sometimes severe headache and epistaxis. Pain in the belly is not uncommon and is more frequent in young children than pain in the chest. It may be referred to the right iliac region. Probably pleural pain is rare, for dyspnœa is much more marked in adults than in children under 5 years of age. Diarrhœa is not infrequent; sometimes there is abdominal distension and constipation. Cough is slight and may be absent for 2 or 3 days or throughout. At the onset it may be painful, short and suppressed, and induce crying. It is most marked in defervescence.

In a few hours the face is flushed, the eyes brilliant, the temperature raised, the breathing rapid, pulse full and frequent, and skin pungently hot. The child passes into a state of drowsiness or stupor; or less often becomes restless and delirious.

The breathing is frequent from the onset but there is no true dyspnœa. It is increased out of proportion to the fever and pulse, ranges from 40-60 or more per minute, is shallow or superficial, and mainly abdominal. The accessory muscles are not brought into action and there is no recession of the lower ribs, for there is no mechanical interference with the entrance of air into the chest. Usually breathing is quiet, but it may exhibit a curious expiratory moan, grunt or pant, possibly due to the pleura being affected. An inverted type of breathing may be seen in infants and young children for hours or days. The pause takes place after inspiration instead of after expiration and the air is momentarily retained in the chest. Sometimes the breathing is of a cerebral type, irregular, cyclical in character, with a temporary slowing or actual pause, somewhat like Cheyne-Stokes breathing. These peculiarities in respiration are perhaps due to disturbance of the respiratory centre in the medulla from stimulation of the peripheral ends of the vagus. Possibly this also accounts for the initial vomiting and the fact that the heart is not increased in frequency in proportion to the respiration. If the vagus is cut and its central end stimulated, the respirations are accelerated and the heart is slowed. This increased frequency of breathing is present from the onset, is out of proportion to the rise of temperature, and does not vary directly as the extent of lung involved. The rate falls with the crisis, still further in another 2-4 days and finally to normal 7-10 days after the crisis. An increase in respirations with a fall or no rise of temperature may indicate cardiac failure or muscular exhaustion. If the temperature rises without any increase in respirations, the fever is not due to extension of lung mischief.



The ratio of pulse to respiration is not as reliable as in adults, for the pulse is so easily accelerated in infants. It ranges from 140-180, and a high rate is not of itself unfavourable. At first the tension is raised.

During the progress of the disease the facial aspect is generally placid, neither anxious nor distressed, in spite of the rapid breathing, unless there is pain. Flushed cheeks are common, but sometimes there is marked pallor and depression. The alae nasi dilate, usually during expiration. This expiratory dilatation is associated with the inverted type of respiration and due to forcible expiration. Active dilatation during inspiration is commonly a sign of inspiratory obstruction. Herpes labialis is present on the second to the fourth day, though frequently absent in infants, especially in the first year.

Pain is usually absent. If it is abdominal it is generally situated in the epigastrium or at the navel, occasionally in the right iliac fossa and associated with muscular rigidity. Cough is short and suppressed and may induce crying because of pain. Sputum can be collected by inserting a tongue depressor to the back of the throat, causing cough and collecting the mucus on a swab. Gastro-intestinal symptoms are common. Anorexia, diarrhoea or constipation, sometimes vomiting, and distension may be present throughout. Jaundice, high temperature and vomiting may be early symptoms. Tympanites is frequent and serious. The knee-jerks may be diminished or absent, but this does not influence prognosis or assist in diagnosis.

The temperature chart is often as typical as in adults. In infants it is frequently irregular, showing diurnal variations of 1-3° F., or even more marked oscillations, especially towards the end of the process. In rare cases oscillations of 3-10° are present throughout. The inflammation of the lungs is not the cause of the fever, for the temperature is often at its highest before any such inflammation is evident. The temperature falls suddenly, though no change can be traced in the condition of the lung, and its height is not proportionate to the amount of lung involved. The fever ends by crisis on the fifth to the ninth day, most frequently on the seventh. Abortive cases may terminate on the second or third day. A temporary fall of several degrees of temperature, sometimes to normal or even below, for a few hours is called a *pseudo-crisis*. It occurs more frequently in the young than in older subjects, usually on the third to the fifth day, and is a good sign. The symptoms are temporarily relieved but become worse with a further rise, which lasts for 24-48 hours and is not a sign of extension. Crisis is least common in infants. Sometimes it does not occur until the fourteenth day or later, because of extension to another lobe, pleurisy or bronchitis. Prolonged cases are more likely to end by lysis. A crisis is generally accompanied by considerable prostration, but rarely by the collapse sometimes seen in adults. The critical discharge is commonly profuse sweat and



the child drops into a quiet sleep. A post-critical fever may last for a few hours. If prolonged, it indicates spread of mischief or a complication.

Up to the time of the crisis the patient steadily gets worse. The tongue is thickly coated and dry, and in 5 or 6 days the mental condition one of apathy, somnolence or delirium. The urine is febrile in character, deficient in chlorides, contains an excess of urates and urea, and sometimes albumin. The patient is generally worst just before the crisis, but afterwards is soon bright and well, or in 2 or 3 days if the attack is severe.

*Physical Signs.*—Examination is often difficult because of restlessness and irritability. Definite physical signs may be slight, delayed until the fourth or fifth day, not present until after the crisis, absent throughout, or missed because they are so deeply seated. Central pneumonia is frequent in children, especially in the upper lobe, and the physical signs may be limited to persistent rapid breathing and high fever.

In basal pneumonia expansion is deficient in the subclavicular region on the affected side. Generally speaking the early signs are distant, faint or absent breath sounds with a tympanitic note due to vicarious emphysema, and harsh puerile breathing on the opposite side. Consequently the sound side seems more dull and the breathing may be mistaken for bronchial breathing. Crepitations are more often absent than present. They may be only heard when the child cries. Vocal vibrations afford no assistance in young children. Bronchial breathing and bronchophony, especially the latter, are most valuable signs and may be heard on crying or coughing, even on the second day in basal pneumonia. In apical pneumonia they are generally heard over the spine of the scapula on the fourth to the sixth day. Inspiration may be mistaken for expiration in the inverted type of breathing, and bronchial breathing will then seem louder on inspiration instead of, as normal, in expiration. Always examine high up in the axilla, for consolidation here is often overlooked. The physical signs clear up with remarkable rapidity, often within 2 or 3 days of the crisis and almost invariably within a week.

*Clinical varieties.*—Certain types are described in accordance with the predominance of particular symptoms—

1. *Cerebral Pneumonia*, sometimes called eclamptic, if fits predominate; meningeal, if coma and delirium. It may begin with convulsions. Headache, vomiting, delirium or drowsiness, and sometimes retraction of the head may be conspicuous features in the early stages and before physical signs can be found in the chest. This type is the “brain fever” of the novelist. Active delirium is rare under 3 years and generally present after the fourth year. It may be wild or muttering and is worst at night. It is generally associated with tremor of the tongue and hands, muscular twitching of the face and limbs, retraction of the head, opisthotonos, general rigidity, intolerance



of light, screaming and crying when disturbed, sometimes unilateral contractures and fits. The child is completely unconscious and the pupils contracted. In other cases the cerebral condition is one of drowsiness as in the typhoid state. Cerebral symptoms are more frequent in apical than in basal pneumonia. They are due to toxæmia, or to hyperæmia, congestion and œdema of the nervous tissues, or may depend on a high temperature. They may be so marked that the chest is not even examined. The physical signs precede, accompany or follow the cerebral ones.

A true meningo-encephalitis may occur as part of the pneumococcal infection and develop at the height of the fever or not until later. It cannot be diagnosed unless the base of the brain is involved. The pneumococcus has been found in the exudate. Sometimes the cerebral symptoms depend on acute otitis.

2. *Pleuro-pneumonia*.—In these cases the pleurisy is prominent and masks the pneumonia. The physical signs depend on the amount of exudation. If there is much, the breath sounds are less distinct, dulness persists long after the crisis, or the case runs a chronic course and ends by lysis.

3. *Gastro-intestinal Pneumonia*.—These cases present the aspect of typhoid fever. The tongue becomes inflamed, anorexia and vomiting are marked, diarrhœa is sometimes severe and intractable, slight jaundice may be present, and the abdomen is distended, hard, and may be tender. Abdominal pain and constipation are more frequent than diarrhœa. Cough may be absent. There may be no physical signs for some days, perhaps not until after the crisis, and in infants even absent throughout. Some of these cases are instances of pneumococcal pyæmia or direct infection of the alimentary tract.

4. *Nephritic Pneumonia*.—In rare instances there is an acute hæmorrhagic nephritis with general anasarca from the onset. It is difficult to be certain whether this or the pneumonia is the primary disease. An acute onset with vomiting, rapid breathing, and high fever is improbable in acute nephritis. The renal symptoms subside rapidly after the crisis, not lasting more than 2 weeks and ending in complete recovery. Nephritis may be distinctly secondary and become subacute, persisting for weeks after defervescence. The true nephritic pneumonia must be regarded as part of the general pneumococcal infection.

5. *Pneumonic or Pneumoccal Fever*.—Cases often occur which would be diagnosed by the symptoms and course as pneumonia if the chest were not examined. Physical signs are absent throughout.

6. *Abortive Pneumonia*.—In these cases the fever lasts only a few days, even one or two, but the signs of consolidation appear later.



*Diagnosis.*—The chief difficulties arise from the absence of a characteristic onset or symptoms, and of sputum, and the presence of abnormal types of disease. The main features are the vomiting at the onset, rapid breathing, high fever, flushed cheeks, dilating nostrils, expiratory grunting, inverted respiratory or cerebral rhythm, short course and critical fall. Intractable vomiting (*pneumonie emetisante*) simulates peritonitis if associated with high fever, abdominal pains and rigidity. Cases with vomiting, pain in the right iliac fossa and rigidity may be mistaken for appendicitis, but the abdomen is not tender and respiration is unduly frequent. The association of fever, gastro-intestinal and nervous symptoms may suggest acute ileo-colitis or enteric fever. Even in the pronounced meningitic type the ratio of pulse to respiration, rapid breathing, high temperature and flushed cheeks enable a diagnosis to be made. Central pneumonia with gastric symptoms simulates acute meningitis. Occasionally cerebrospinal meningitis is ushered in by fits, high fever and rapid shallow breathing indicative of a serious toxæmia impossible of diagnosis from pneumonia. So, too, acute specific fevers cannot always be differentiated at the onset. The differential diagnosis between true pneumonia and primary broncho-pneumonia depends on a careful consideration of the course and progress of the disease and is often impossible. The diagnosis of double pneumonia from bilateral broncho-pneumonia is also very difficult. Occasionally acute empyema closely simulates pneumonia.

*Prognosis.*—Cases are rarely fatal when sthenic and uncomplicated. The mortality in childhood ranges from 2-5 per cent., and is extremely small after the second year of life. Death may result from pneumococcal meningitis; acute otitis and secondary thrombosis of the lateral sinus, meningitis or cerebral abscess; purulent pericarditis or pleurisy; or from toxæmia. The toxin acts upon the heart muscle and causes dilatation, and on the cardiac or vasomotor centre in the medulla. Death is sometimes sudden and due to one or both of these causes. The heart stands the strain well, especially in infancy, for the right ventricle is strong and dilatation is uncommon. Fatal syncope in convalescence is very rare.

The prognosis varies with the age and nutrition. Wasted infants die. Girls have less resisting power than boys. Rickets and pre-existing heart lesions or lung affections increase its gravity. The nephritic type is sometimes fatal. Convulsions at the onset are of no prognostic value. They rarely occur after the second year. During the course of the disease they usually indicate a fatal issue. The sthenic cases show the most extensive lesions and the greatest fever. No estimation of the extent of the lung involved or the duration of the illness can be based on the violence of the onset, the height of the fever, or the severity of the nervous symptoms. The greater the extent of the lung involved, the worse is the prognosis. The outlook is good if the temperature is under 103° F., bad if it is over 106° F. and serious if the fever is prolonged beyond 9 days. It is very fair as long



as the pulse remains under 150 and the breathing under 60 per minute. Diarrhœa and pericarditis are serious complications. Tympanites is a bad sign. Acute abdominal distention is not an uncommon precursor of death. A high leucocytosis may indicate a powerful reaction or virulent infection.

*Complications and Sequels.*—In spite of the blood infection complications are rare. Dilatation of the right auricle must be examined for daily. Empyema is the most important sequel and gives rise to the usual signs of fluid in the chest, persistent fever, or a secondary rise shortly after the crisis. Thick creamy pus may be found a day or two after the crisis, although loud bronchial breathing is present. Otitis media and broncho-pneumonia are quite frequent. Among the other complications may be mentioned hyperpyrexia, endocarditis, pericarditis, peritonitis, meningo-encephalitis and secondary chronic hydrocephalus, post-pharyngeal abscess, jaundice, acute nephritis, purulent arthritis, osteomyelitis, abscess in the lung, gangrene due to food aspiration, and subcutaneous emphysema. The pulse during convalescence may be infrequent and very irregular, as a sequel of the action of the toxin on the cardiac muscle.

*Treatment.*—The natural tendency of pneumonia is to recovery. Keep the child in bed, lightly clothed, in a well ventilated room with open windows and temperature 60° F. Have an oxygen cylinder handy if there is much cyanosis. Tent beds, steam and antiseptic vapours are rarely advisable, but towels soaked in creosote and eucalyptus may be hung up in the room.

Give water freely, especially if the child refuses other fluids; or a plentiful supply of weak nutritious digestible foods, e.g., milk, whey, weak tea, fruit juice, plasmon, meat juice, broth and eggs. A healthy well-nourished child can do quite well without food for 24-48 hours during the early acute stage. Overfeeding may cause acute abdominal distension, vomiting and diarrhœa. Do not wake the child for food unless it is very weak. It must be fed nasally if necessary. Alcohol is rarely needed before the crisis.

Locally apply poultices, mustard leaf, ice bag or ice poultices, or leeches. Heat is better borne than cold and children commonly resent ice bags. It is not always clear which portion of lung is affected, and there is no proof that local applications of cold affect the underlying lung or that the treatment is beneficial, even if it does affect the lung. A mustard leaf or intermittent turpentine stupes relieve the pain. Dry cupping and leeches are even more efficacious. A pneumonia jacket is of doubtful advantage.

In an ordinary simple case give an initial dose of calomel or saline cathartic, and a mixture of citrate and acetate of potash, spirit of nitrous ether and camphor water every 3 or 4 hours. Tepid sponging of the skin is comforting to the child, and helps to reduce the fever. If there is much vomiting and constipation, small doses of calomel can be given frequently. The mouth and teeth must be kept clean.



Antipyretic measures are more necessary for nervous symptoms than high fever only. Apply ice to the head. Treat fever and hyperpyrexia according to the measures recommended on p. 90.

Expectorants do more harm than good. Drugs such as *tr. veratriæ* and *tr. aconiti*, phenazone and other coal tar derivatives are depressants. Small doses of phenacetin can be given if the temperature is very high, and to allay headache, restlessness and nervous irritability. *Digitalis*, in my opinion, is more likely to do harm than good, unless arterial tension is very low and the pulse feeble and weak. In older children, and to a less extent in the very young, the common condition in pneumonia is one of high arterial tension with a feeble and distended right auricle and ventricle. Heart failure is due to the action of the toxin or prolonged high temperature on the cardiac muscle, for which *digitalis* is not advisable. A few leeches over the liver will relieve dyspnœa, distress and insomnia due to dilatation of the right heart, by reducing the size of the auricle. It affords relief for one or two days, can be repeated, and is not necessarily contra-indicated by pallor of the face and smallness of the pulse. After leeching it is possible that *digitalis* or *strophanthus* may be useful, but it is better to rely upon *strychnia*, *nux vomica*, alcohol, *caffein*, ether and ammonium carbonate. *Strychnia* is of more value than in adults to counteract the easily depressed nerve tone, and it must be given freely if acute tympanites develops. Cardiac tonics are often wanted before the crisis and quinine is one of the best.

Severe dyspnœa and cyanosis are treated by oxygen inhalation and mustard poultices, dry cupping of the bases of the lungs or leeches. The cyanosis is often due to cardiac dilatation, but bleeding is rarely necessary, except by leeches over the liver in childhood. For toxæmic dyspnœa give atropin and *strychnia* subcutaneously. For convulsions give chloral per rectum. Troublesome and irritating cough is treated by change of posture, inhalations, small doses of *vinum ipecac.*, paregoric, heroin, oxymel *scillæ*, syr. of tolu, and such-like remedies. A small dose of chloral, ammonium bromide, or urethane may be added. The latter drugs are useful for sleeplessness and delirium and preferable to Dover's powder or morphia, which are sometimes necessary. These symptoms are best treated by cold to the head, tepid sponging, food and alcohol, rather than by sedatives. Sponging with cold water, a drink of milk, and a dose of bromide or veronal is generally efficacious. If the cerebral symptoms are marked, give calomel freely. Serum treatment is of little value.

Three days after the fall in temperature the child may be allowed out of bed, if the lung signs have cleared up and there is no cardiac dilatation. In another week he may go out. Tonics, liberal diet and general care are required during convalescence.

**Chronic Pneumonia.—Bronchiectasis.**—Chronic inflammation of the lung is generally tuberculous. It may occasionally follow pneumonia,



broncho-pneumonia, pleurisy, foreign bodies in the bronchus, or pressure by intra-thoracic glands. It is especially apt to follow the infective diseases which give rise to broncho-pneumonia, and is therefore most frequent at 2-3 years of age.

The affection is a chronic interstitial fibrosis of varying causation and distribution. There may be a general firm adhesion of the lung, one lobe or portion of a lobe to the chest wall, diaphragm and pericardium. On section, the affected lung is found hard and fibrous, with more or less complete disappearance of alveolar structure, and traversed by dilated bronchi. If secondary to bronchitis or broncho-pneumonia the fibrous changes are more patchy in distribution, surrounded by emphysematous areas; and the chronic inflammatory process appears, microscopically, to have started and to be most advanced round the dilated small bronchi and bronchioles.

*Bronchiectasis* or *bronchiolectasis* is a dilatation of the large or small bronchial tubes, the result of atrophy of their walls and subsequent thickening from peribronchitis. There is always more or less evidence of a basement membrane. It may follow or precede chronic pneumonia, and the two affections cannot be clearly differentiated. It is unilateral or bilateral. It may affect both lungs, one lobe or lung, both lower lobes, and rarely both upper or the right upper and middle lobes. Like pneumonia it is most frequent in the left lower lobe. Its etiology is the same as that of chronic pneumonia. Sometimes it can be traced to a chronic cough or chronic pleurisy.

Three forms have been described. (1) Simple, cylindrical or spindle-shaped; (2) Saccular; (3) False or trabecular bronchiectasis. The cylindrical variety is an acute dilatation of the tubes, due to acute bronchitis and whooping cough. Chronic bronchiectasis is partly cylindrical and partly saccular, and shows considerable fibroid thickening. The lung is eventually composed of fibroid tissue and dilated tubes. *Cystic disease of the lung* is a congenital variety of bronchiectasis, usually bilateral, in which the lung is cystic throughout.

The cavities are multiple, more or less spherical, and in size up to that of a pea. Sometimes they are larger and very irregular in shape. They are empty or contain purulent secretion, and may communicate with each other. Their walls are thickened and they are usually separated from each other by bands or septa. There is much fibrosis and destruction of alveoli round the cavities and patches of consolidation. The adventitia of the blood vessels is thickened.

Some are undoubtedly dilated bronchioles, for they are lined by columnar epithelium and contain muscle and elastic fibres in their walls. Others are dilated infundibula and atria, or due to the discharge into bronchioles of softened pneumonic patches. Some of these may project, as bullæ, above the surface, if the pleura is non-adherent.



Various factors are concerned in their production. The resistance of the walls is diminished in bronchitis and general malnutrition. A distending force is exerted from within by violent cough, and by retained secretion in the case of foreign bodies. Traction on the walls from without is exerted by contraction of fibrous tissue in the lungs and pleura. According to the theories of their formation, some of the cavities are due to necrosis of lung tissue, forming minute abscesses that communicate with the bronchioles. Others are due to a true bronchiolectasis from dilatation of the terminal bronchioles and infundibula; these cavities not projecting above the surface of the pleura. In a third type, *atriectasis*, the atria of the bronchioles are dilated and several infundibula open into one atrium. Probably all three forms are present in a single case, except in the acute cylindrical type of the disease.

*Symptoms.*—Sometimes there are no signs or symptoms, the lung inflammation has subsided and death is due to intercurrent disease; or the symptoms are those of chronic bronchitis. Other cases present the physical signs of disseminated broncho-pneumonia, and a “honey-comb” lung is found after death. In a fourth type the cases are of prolonged duration and give rise to the characteristic signs and symptoms.

In a case which follows lobar pneumonia, with or without pleurisy, the temperature falls by crisis and remains down, or comes down gradually to about the normal level and the physical signs do not clear up. There remain persistent dulness, rales and patchy bronchial breathing, and general feebleness of air entry. The dulness may increase and the signs of bronchiectasis develop. This condition may persist for months with little variation, or be modified by recurrent attacks of bronchitis or broncho-pneumonia, each attack leaving the lung a little worse than before. The child is pale and wasted, but may subsequently gain strength and flesh and appear robust, although the physical signs do not disappear and a variable degree of cough persists.

In a well-marked case the face is pale and puffy, sometimes congested. Dyspnoea is present on exertion and may be obvious even at rest. Breathing is rapid and shallow. The temperature is normal or raised, nutrition is well maintained, and there are no night sweats and no tubercle bacilli in the sputum. The cough is troublesome and paroxysmal, especially in the morning and at bedtime, and often induced by change in posture. It is aggravated by fresh catarrh. The breath is offensive and expectoration copious. The sputum consists of tenacious, airless, greenish pus, rarely foetid in children, and separates into three layers on standing. It is coughed or vomited up in considerable quantity once or twice a day. Hæmoptysis is not infrequent. Old standing cases exhibit clubbing of the fingers and toes, slight cyanosis, emphysema of the opposite lung, and displacement of the heart to the affected side.



The physical signs vary with the amount of secretion in the cavities. If there is much secretion, the breath sounds are feeble or absent, the note dull, the vocal vibrations diminished, and the vocal resonance indistinct. If the cavities are emptied by cough, the breath sounds are louder, the note more resonant, vocal vibrations increased, and sharp rales, bronchial breathing, bronchophony and pectoriloquy are present. The shadow with X-rays becomes less dense.

*Diagnosis.*—It is most difficult to exclude tuberculosis. Limitation to an upper lobe is more likely to be tuberculous than if the disease is in the lower lobe. Chief stress must be laid upon the absence of tubercle bacilli, the good general nutrition, the very slow course tending to improvement, the absence of further breaking down of lung tissue, the feeble air entry and the absence of fever. Hæmoptysis may occur in either disease. A more common error is to mistake an empyema discharging through the lungs for bronchiectasis, or simple empyema following pneumonia for chronic pneumonia. Some cases suggest new growth; but in course of time this can be excluded by the absence of an increase in the area of dullness and of cardiac displacement to the opposite side.

*Course and Prognosis.*—Fœtid expectoration and fever are bad signs and indicative of constitutional disturbance due to toxic absorption. Acute bronchiectasis from bronchitis or pertussis almost invariably gets well, unless death results from the primary disease. Chronic cases can be arrested, improved by treatment and may get well. Many are more or less progressive and incurable, but last for years. The prognosis in the very young is better than generally supposed. It depends on the cause, the state of the surrounding tissues and the social environment, and varies directly as the extent. A limited area is harmless. A more extensive one interferes with general health, and causes dyspnœa on exertion. Many children remain permanently delicate and phthisis is not an uncommon termination. Some die from broncho-pneumonia. Nevertheless a severe case, involving a whole lower lobe, may improve to such an extent that there is little evidence of mischief in a year's time.

*Complications.*—Recurrent inflammation, bronchitis and broncho-pneumonia are common. Septic absorption leads to hectic fever and wasting. Other complications are hæmoptysis, phthisis, enlarged and perhaps caseous bronchial glands, fatty and amyloid degeneration, pulmonary abscess, gangrene, pneumothorax, subcutaneous and mediastinal emphysema, suppurative meningitis, cerebral abscess, cardiac failure, nephritis, periostitis and arthritis.

*Treatment.*—Prevention consists in the proper treatment of the initial disease, notably of broncho-pneumonia in the course of measles and whooping cough. Subsequently it must be directed to emptying the cavities, relieving fœtor and encouraging the full expansion of the lungs. The child's general health must be maintained and guarded against catarrh.



The cavities can be emptied by inversion of the body, holding the child up by the legs and compressing the chest or abdomen during expiration, or by leaning the body over the side of the bed with the hands on the floor. It should be done three times a day. Give expectorant mixtures containing iodide of potassium, and an emetic of vin. ipecac. every fourth day if the child is strong enough.

Prevention of foetor wards off septic complications for it is due to bacterial decomposition. It is relieved by spray producers, atomisers or inhalers with terebene, ol. pini. sylvestris, eucalyptus, creosote, iodine, tar compounds, carbolic acid or thymol; by creosote vapour baths; by intra-tracheal injections of izal, 5 per cent. in glycerin, or guaiacol 1, menthol 5, olive oil 94 parts; by the internal use of deodorising and antiseptic drugs given in capsules after food, e.g., creosote, oil of garlic, syr. allii U.S.P. or syr. picis liq. U.S.P. t.d.s. A suitable mixture consists of ol. tereb. dr. 1, mellis oz.  $\frac{1}{2}$ , pulv. trag. co. oz.  $\frac{1}{2}$ , aq. menth. pip. ad oz. 3, in doses of 1 dr. t.d.s. Balsams and sulphur waters are also useful. Choose for these patients a dry, warm, equable climate such as Jamaica (November to April), the Mediterranean, South California, South Africa and the West Indies. The general treatment is that of phthisis; good hygiene, liberal diet, respiratory exercises and tonics. Operative treatment is inadvisable, as there are many cavities. Gluck's operation, extirpation of part of the lung, was first done in 1885. The middle lobe was removed successfully by Korte (1907) in a boy aged 13.



## CHAPTER XXXIX.

### THE PLEURA AND PLEURAL CAVITY.

*Hydrothorax—Multiple Serositis—Pleurisy—Pleural Effusions—Empyema—Pneumothorax.*

There is a continual circulation of lymph through the pleural sac, via the stomata and lymphatics. It is maintained by respiration, and absorption plays a very small part in the process. During inspiration fluid is sucked in and on expiration it is driven out. Just enough fluid is kept in the sac to prevent friction. In hydrothorax it enters more rapidly than it can be removed. In inflammation, exudation is increased and the stomata are blocked by congestion, pressure and exudation, thus preventing its removal. With increasing effusion the respiratory movements become more limited and the effect of expiration in getting rid of the fluid is reduced. In large effusions the respiratory movements on the affected side may be entirely abolished and no re-absorption can occur. The removal of a small portion of the fluid sets in action the respiratory movements and leads to disappearance of the fluid, an effect not infrequently seen in practice. Treatment by limitation of fluid ingesta, purgation, diuresis and diaphoresis is illogical and useless for it cannot affect the mechanical state of affairs in the chest. It may prove of some value in hydrothorax from nutritional changes.

**Hydrothorax** is due to mechanical causes, nutritional changes in the endothelium and altered conditions of the blood. It is not common in children but is occasionally met with in obstructive heart or lung disease, renal affections, new growths and blood states such as leukæmia.

**Multiple Serositis**, also called *polyserositis*, *polyorrhomenitis* or *Concato's disease*.—This is a general inflammation of the serous membranes, with effusion into the pericardial, pleural and peritoneal cavities. Strictly speaking it is not due to tubercle or new growth. Some of the cases described under this heading are tuberculous in origin; a few possibly of rheumatic origin; and others are associated with hepatic cirrhosis, chronic interstitial nephritis or indurative mediastinitis. Occasionally the effusion is purulent, and there may be a collection of pus behind the parietal pleura, adjacent to the spine, due to extension of the mischief along the lymphatics.

**Pleurisy** is divisible into the dry or fibrinous variety, and pleurisy with effusion which may be serous or serofibrinous, seropurulent or purulent



(empyema), or hæmorrhagic. Hæmorrhagic effusions in children are not necessarily due to tubercle or new growth.

Apart from this last variety the etiology of these affections may be considered together. They may occur at any age. In the newborn they are commonly due to sepsis. In a stillborn infant, born at full time without difficulty, I found recent dry pleurisy with no other pathological lesion. In early life inflammatory effusions are commonly secondary to inflammatory or zymotic disease and have a marked tendency to be purulent. Most of them are secondary to pneumonia, broncho-pneumonia, tuberculosis and hæmorrhagic infarction of the lung; to infectious diseases such as measles, scarlet fever, typhoid fever, diphtheria and influenza, usually secondary to lung mischief; to pyæmic affections such as sepsis, appendicitis and osteomyelitis; to rheumatism; to extension from adjacent organs and to new growth. The disease almost always is a sign of a general morbid state and not merely a local affection. It generally indicates lung mischief and is due to the pneumococcus or the tubercle bacillus. Of these the pneumococcus is far the more prevalent in early life, and consequently pleural effusions in the young are generally purulent. About 90 per cent. of cases of empyema are secondary to lung mischief. A few are primary or synchronous with pneumonia. All varieties of effusion are more frequent in the spring months, and rather more common in boys because of their greater liability to pneumonia. Practically two-thirds of 300 collected cases of empyema occurred under 5 years of age, and one-third between 5-10 years.

*Morbid Anatomy.*—In dry pleurisy the amount of lymph varies from merely enough to cause pain and friction to a thick layer covering an extensive area of lung. Its appearance varies with the number of pus cells. There may be only a few fibrinous adhesions, loss of lustre, and roughening of the pleural surface; or a thick yellowish or yellowish green layer of lymph. The latter is present in pleuro-pneumonia. In tuberculous pleurisy grey or yellow tubercles are present and a moderate exudation of serum and lymph, except in those cases which have become purulent from secondary infection with the pneumococcus or pyogenic organisms. In empyema the exudation of pus is preceded by that of lymph and the consistency of the fluid depends on the relative proportions of the serum and pus cells. The extent of the fibrinous exudation is generally greatest in pneumococcal infections and large masses of lymph are found covering the pleural surfaces. Thin yellowish seropurulent effusions are often due to a streptococcus, and thick pale green ones to the pneumococcus, but the naked-eye appearances are unreliable evidence of the type of organism present. Empyemata are primary effusions. Occasionally a serous effusion becomes purulent in course of time. It is sometimes unfairly stated that serous effusions do not become purulent except from lack of care in exploration. There is no doubt that this is untrue and that secondary



infection with the pneumococcus or pyogenic organisms does occur. In pneumonia the effusion may be serous at the onset and purulent at a later date. Sometimes a bilateral effusion is serous on one side and purulent on the other, and a multilocular unilateral empyema may contain some loculi filled with serum.

Both serous and purulent effusions may be localised, shut off by adhesions ; or sacculated, in several pockets ; or general, due to adhesions being broken down by the accumulated fluid. They may be situated in any part of the chest, most often posteriorly at the base. A localised serofibrinous effusion after infancy is usually over the right middle lobe. Localised empyemata are found in the large fissure of the lung, between the base and the diaphragm, or between the vertebral column and the lung, and are very rarely apical. In pneumonia it is by no means uncommon to find a small localised effusion of thick creamy inoffensive pus, perhaps amounting to barely 1 dr. of fluid and hardly deserving the name of empyema.

*Examination of the Fluid.*—Withdraw some of the fluid from the chest, centrifugalise it gently and examine the deposit under the microscope (Cyto-diagnosis). In hydrothorax only a few flat endothelial cells are found. In recent tuberculous pleurisy the cells are almost entirely small, mononuclear lymphocytes. Sometimes in early stages of tuberculous pleurisy there is an excess of polymorphs. This probably means primary pleurisy and secondary tuberculous infection, or a secondary infection of a tuberculous one. In tuberculous cases of long standing, in non-tuberculous infective pleurisy, and in cases becoming purulent or complicated by pneumonia, the polymorphs are much increased in number and are in great excess.

*Jousset's Method* is used for finding tubercle bacilli. Draw off a few ounces of fluid and allow it to clot. Digest the clot with artificial gastric juice, again centrifugalise and stain the precipitate. The tubercle bacilli are entangled in the clot, unaffected by the digestion, and are precipitated.

Cultural and inoculation methods afford positive results in many tuberculous cases, but leave others doubtful and take a long time. The tuberculin test is available in apyrexial cases and only indicates a tuberculous process somewhere. Eosinophilia is of no diagnostic importance.

*Bacteriology.*—Serous effusions are commonly due to the pneumococcus, especially in nurslings, the streptococcus, staphylococcus and tubercle bacillus. Idiopathic pleurisy with effusion is more often tuberculous than commonly supposed. Empyemata are due to the pneumococcus alone in 60-90 per cent., the streptococcus alone in 5-15 per cent., the tubercle bacillus in about 5 per cent., while the remainder are due to mixed infections. Widal and Philibert (1907) have shown that some pleural effusions, in which the pus is microscopically like that of pneumococcal pyæmia, may be sterile



and contain intact polymorphs. It is much more difficult to determine the responsible organism in serous than in purulent effusions.

The general conclusions are that the pneumococcus is the most common cause of empyema, especially in infants; that the disease is usually secondary to lobar pneumonia; that pyogenic germs may be found associated together, or with the pneumococcus, and are chiefly present in cases of pyæmic origin and infective disease; and that in tuberculosis there is usually a secondary infection. A sterile empyema is almost certainly tuberculous.

*Symptoms.*—Pleurisy gives rise to pain in the side, worse on drawing a deep breath and on cough. The pain may be severe and is sometimes referred to the umbilicus or the right iliac region. Except in tuberculous cases the onset is sudden, but rarely as acute as in pneumonia. The disease is ushered in with fever and chilliness, headache and general malaise, sometimes vomiting. The temperature rises to 101-103° F., occasionally higher; pulse 100-150; and the breathing is frequent and shallow, with short, hacking, dry, restrained cough. There is no dyspnoea or cyanosis. The affected side may be tender. Usually the child lies on its back or, if there is no local tenderness, on the affected side, so as to limit the respiratory movement on that side and allow free expansion of the opposite lung. Friction and perhaps a little impairment of resonance are found on examination. If there is much lymph it gives rise to marked dulness, weak breathing, and deficient vocal vibrations and vocal resonance.

A baby with pleurisy is very ill and looks still more so. The eyes are sunken, the aspect suffering, and the skin somewhat bluish. The temperature may suggest pneumonia. Breathing is shallow and consequently there may be no friction sounds. Sometimes there is impaired air entry and distress on movement. If the pleurisy is diaphragmatic the attack may suggest peritonitis, for the legs are drawn up and the upper half of the abdomen is immobile. Breathing is still more shallow, frequent, and often irregular and apparently painful. Should the attack be on the right side pain is caused by pushing the liver upward. A little impairment of resonance is found at the base and in the axilla.

The *course* of dry pleurisy is either that of the disease with which it is associated or is quite short, lasting only a few days to a week. Some adhesions are left. In severe cases with extensive thick exudation the whole pleural cavity may be obliterated and the lungs become universally adherent to the chest wall. A thickened pleura may be left, causing impaired resonance and deficient expansion of the lung.

Simple pleurisy is rare in early life except as a complication, generally of bronchitis, pneumonia or broncho-pneumonia. Herpes zoster, myalgia, neuralgia, and local inflammatory affections may cause similar pain. True pneumonia must not be overlooked. Weak breath sounds and absence of bronchial breathing are more valuable signs than the degree of dulness and alterations in vocal vibrations and vocal resonance.



By the efficient *treatment* of pleurisy it is possible to prevent the effusion of fluid. Keep the patient in bed and adopt the ordinary treatment of febrile states until the local signs have cleared up.

Severe pain is relieved by counter-irritation, mustard leaves, hot fomentations, etc., or strapping with adhesive plaster. Leeches are useful in older children. In babies the pain is best relieved by opium. The general treatment is that of fever. Absorption of thickened pleura may be aided by painting with tincture of iodine and pot. iod. internally.

**Pleural Effusion.**—There is generally a history of pain in the side or some lung trouble followed by gradually increasing shortness of breath, varying in degree with the amount of fluid present and increased on exertion. Sometimes there is an intervening period of a few days of apparent health. Both in pleurisy and pleural effusion the pupils are not infrequently unequal, the one on the affected side being a little dilated and sluggish in reaction to light. This is probably due to peripheral irritation of the sympathetic.

The fluid exerts mechanical effects on the heart, mediastinum, lungs and diaphragm. Consequently the symptoms are increasing dyspnoea and frequency of the pulse, with a variable amount of general malaise and perhaps fever. The physical signs are those of cardiac displacement, collapse of the lung and the presence of fluid.

As the fluid increases in quantity the lung becomes more and more compressed, until it is eventually quite collapsed and airless and is withdrawn into the angle formed by the ribs and the vertebral column. Its elastic traction is reduced and then abolished, so that it cannot counteract the elastic traction of opposite lung. Hence the mediastinum and heart are *drawn* over to the opposite side, the cardiac apex is displaced and the area of dulness altered; and the diaphragm is depressed. These effects are most marked in left-sided effusions. Dulness and pulsation are found to the right of the sternum, pulsation is present in the epigastrium, and the heart may be felt beating beneath the right nipple. The position of the apex must be carefully located with the child lying on the back, and any alteration from day to day must be noted. An apparent initial displacement to the left is of little value, for it may be due to cardiac dilatation or be the normal position in infancy and early childhood. Alteration in the position of the heart, occasionally so marked as to suggest transposition, causes breathlessness, often a systolic displacement murmur, and a liability to syncopal attacks.

The more the lung is compressed the greater is the dyspnoea, but the child quickly accommodates itself to altered conditions if the fluid effuses slowly. In rapid effusions the distress is very great, while in slow ones it may be so slight as to attract no attention. The amount of fluid varies with the age, size of the child, and the duration and character of the



effusion. It is greater if the fluid is thin. Under 3 years of age there is about  $\frac{1}{2}$ -1 pint, in older children up to 3 or 4 pints.

In early stages there may be some narrowing of the intercostal spaces, from reflex contraction of the intercostal muscles. Later these spaces bulge and the affected side as a whole is expanded, assumes the position of complete inspiration and measures more by cyrtometer. Its movements are deficient. The degree of dulness depends on the amount of fluid and fibrinous exudate. It may be limited to one base, or reach the second rib or higher. It then extends on to the sternum and perhaps  $\frac{3}{4}$ -1 inch to the opposite side of the manubrium, from distension of the pleural sac. The dulness is more wooden and more superficial than in pneumonia. It varies with the size of the effusion and the position of the patient. A small effusion occupies the lateral costo-diaphragmatic furrow if the patient sits up, and gives rise to dulness in the axillary line in the lower part of the chest, with an almost horizontal upper border and a lower border convex upwards. In the dorsal decubitus the fluid occupies the costo-vertebral furrow and the lateral dulness disappears. Larger effusions extend laterally and anteriorly. In the seated position the upper limit in the anterior and lateral regions is horizontal. Behind, at a distance of 1-3 ins. from the vertebral column, the line of dulness falls abruptly, forming a triangle in which resonance persists below the general level of dulness. Sometimes a semilunar zone of impaired resonance, known as "the Hat," is found above the level of dulness, and is due to partial collapse of the lung from pressure. In moderate effusions the anterior level of dulness becomes lower, sometimes higher, in the horizontal posture. In large effusions there is no change with alteration in posture.

*Grocco's Triangle* (1902), or the paravertebral basic triangle of dulness, is a patch of partial dulness adjacent to the spine on the side opposite the effusion. It is not invariably present. The triangle is formed by the vertical line of the spinal processes, the lower limit of pulmonary resonance, and a hypotenuse which may show a slight outward convexity. The apex of the triangle is rather below the level of the upper limit of effusion. It is absent when the patient lies on the affected side, unless the effusion is very large, and present both in the upright posture and when lying on the sound side. This patch of impaired resonance is due to displacement of the mediastinum, conduction of dulness from the opposite side, or to the fluid against the bodies of the vertebræ suppressing bone vibration. It is most marked in right-sided effusions and it is absent in pneumonia or, if present, it does not vary in position.

Dulness may assume abnormal forms as the result of adhesions. Vocal vibrations are diminished or absent, but the sign is difficult to elicit in children because of the character of the voice. Breath sounds are weak and distant, or occasionally absent. Distant bronchial breathing and bronchophony are common, but sometimes the sounds are as loud as over



solid lung, especially near the spine. Nasal voice sounds and ægophony may be heard at the upper limit of dulness. Friction may be heard above the fluid and during absorption. The weakness or absence of breath sounds and vocal resonance are better guides than dulness to the amount of effusion, for dulness may be due to fibrinous exudation. At the apex of the lung the note is impaired, boxy or tympanitic, and bronchial breathing may be present, if there is a great amount of collapse.

Examination by the X-ray screen is not reliable for small effusions at the base of the lung. In larger ones it may reveal a dark shadow with a straight upper border, flattening of the diaphragm, and exaggerated movement on the opposite side. The dome shape of the diaphragm is due to the elastic traction of the lungs. In most effusions the amount of fluid is not enough to create a positive pressure within the pleural cavity. During the process of syphonage to evacuate the fluid, as soon as the opening of the tube is held at the same level or even a little below that in the thorax, the flow is stopped or reversed. In large effusions there may be positive pressure with accentuation of the displacement of various organs. The liver and spleen may be felt per abdomen, through flattening of the diaphragm, and in cases of positive pressure may be actually pushed down.

**Empyema.**—The past history of a case of empyema is that of an acute illness with consolidation of the lung and effusion of fluid. Often the antecedent illness is vaguely described and has not been followed by complete convalescence; or an attack of pneumonia is followed by a fall of temperature to normal or nearly so, and a gradual rise with increasing symptoms instead of convalescence. The lung may not have resolved. In other cases the lung undergoes resolution and apparent convalescence is followed by a gradual development of empyema 3-5 weeks later. Bronchopneumonia is a more common antecedent than lobar pneumonia. Sometimes the history is limited to increasing anæmia, debility, breathlessness and perhaps cough.

*Acute primary empyema* simulates pneumonia. The onset is sudden and symptoms are severe, fever high, and in a few days the side is full of pus, sometimes mixed with blood. There is much dyspnoea. Delirium is earlier in onset and more marked than in pneumonia. It is generally pneumococcal.

*Latent empyema* in infants is often overlooked, because of the small size and slight pulmonary symptoms. Occasionally one cavity is opened but a second one is shut off by adhesions. A small empyema leads to progressive wasting and anæmia with irregular fever and few respiratory symptoms. It is gradual in onset and prolonged in duration. The physical signs may be limited to slight impairment of resonance, generally at the extreme base near the spine and modified in character by the stomach and liver. The signs may vary from day to day. It may give rise to



broncho-pneumonia, purulent pericarditis, meningitis, peritonitis, arthritis and pyæmia. Cases are usually mistaken for tuberculosis or marasmus.

*Interlobar empyema* may consist simply of a layer of pus between the two lobes. *Pulsating empyema* is almost always on the left side, the pulsation being transmitted from the heart or aorta. An empyema may point within a few days or not for weeks; generally in the fifth space in the neighbourhood of the posterior or anterior intercostal membrane, most often in front.

After a purulent effusion has existed for some time it produces certain signs of the presence of pus. The child becomes more and more anæmic, wastes, loses appetite and strength, perhaps sweats profusely, and may have an irregular or hectic temperature. The temperature chart is unreliable and variable. Usually the fever ranges from 100-102° F., and it may be hectic or absent entirely. In still more prolonged cases the clinical picture suggests tuberculosis because of emaciation, fever, clubbing of the fingers, diarrhœa, albuminuria and even general œdema. The abscess may point through the chest wall; burst through the lung, œsophagus or bronchus; or track in various directions, e.g., through the diaphragm and among the abdominal muscles, between the external and internal oblique, or down the spine and simulate psoas abscess.

*Diagnosis.*—We have to determine the presence of fluid, its situation and physical effects, its characters and its causation. The diagnosis of fluid depends on the displacement of the heart, the descent of the diaphragm, the area of dulness, the wooden percussion note and sense of resistance, weak breath sounds, distant bronchial breathing and bronchophony. Grocco's sign, when present, is of much value. The absence of rales and the presence of friction and nasal voice sounds at the upper level of dulness are important signs. The more extensive the dulness, the greater is the probability of fluid, and its extension on to or beyond the sternum is practically diagnostic. The presence of fluid can generally be determined without having recourse to exploration. This should be the last measure adopted.

In infants and the very young it may be impossible to make a diagnosis from the physical signs. They were completely absent in a baby although pus was found on exploration, carried out because of the dyspnœa and general symptoms, and several ounces were evacuated. Occasionally bronchial breathing may be very loud and the signs may be those of the consolidation with which it is often synchronous. Effusion has chiefly to be distinguished from pneumonia, broncho-pneumonia, tuberculosis, fibrinous pleurisy, pericardial effusion, bronchiectasis, subphrenic abscess and new growth. The disappearance of a paravertebral triangle of dulness indicates that persistent dulness is due to thickened pleura rather than continued effusion. Diaphragmatic hernia may give rise to similar physical signs.

*Differential Diagnosis between Serum and Pus.*—A certain amount of importance can be attached to the age of the child, the causation, the



mode of onset, duration and degree of illness. Serous effusions are much less frequent than purulent ones in the first three years of life. After that age the serous effusion rather preponderates. An acute onset is in favour of serum, while empyema comes on more gradually as a complication of pneumonia. Some acute pleural effusions are purulent from the start, and in tuberculous pleurisy the onset is frequently insidious. If due to simple chill or rheumatism, and in the absence of pulmonary disease, the effusion is probably serous. The longer the duration of the illness and the greater the effect on the general health, the greater is the probability of pus. Rigors and sweating are more frequent in purulent than in serous effusions. The physical signs are of little value unless there are bulging, redness, tenderness and œdema. The temperature affords no reliable evidence. It may be strongly suggestive of pus although the effusion is serous. Empyema must be suspected in cases diagnosed as unresolved pneumonia; in pneumonia followed by a rise of temperature, pulse and respiration after an apparent crisis or if they do not fall within a reasonable time; if there is undue delirium on the second or third day, great oscillations of temperature, and tenderness in the affected side.

Small loculated collections can only be differentiated by exploration and the general character of the symptoms. A local collection of serum gives rise to physical signs but no general malaise.

Frequently the differential diagnosis can only be made by *exploration*. Though almost invariably harmless it is not devoid of risk. Strict aseptic precautions must be adopted. Use a syringe about the size of an ether syringe with a needle of medium calibre 2-3 ins. long. Choose a dull area and insert the needle vertically to the chest wall through the lower part of the intercostal space, first drawing the skin a little upward. The child should be on its back, close to the edge of the bed or table, with the arm elevated. Be careful that the needle enters the pleural cavity and does not penetrate the lung. With a little suction enough fluid can be obtained for examination. Withdraw the needle sharply and cover the puncture with a pad of gauze and collodion.

I have never seen any evil effect from exploration. It may cause puncture of the intercostal artery and hæmorrhage into the pleural cavity; puncture of the lung and hæmorrhage or pneumothorax; puncture of the heart or pericardium, if too long a needle is used for exploring the left side, and if empyema has been diagnosed instead of purulent pericarditis or pneumonia with dilated heart. A few cases have been recorded of death occurring very rapidly after puncture of the lung in cases of chronic fibroid disease explored for supposed pus. In some of these hæmorrhage has been profuse and in others slight or absent. Sometimes much frothy mucus is expectorated. Death is due to inhibition of the cardiac and respiratory centres, cardiac inhibition by vagus irritation, or suffocation through bleeding into the air passages. Dangerous symptoms have been followed by recovery at the time and death a few hours later.



*Course and Prognosis.*—Acute serous pleurisy is rarely fatal. It runs a fairly definite course. For a few days the child is ill and feverish. The amount of fluid increases with variable rapidity, then remains stationary and finally disappears. The duration is about 3 weeks. If fluid is left after that, it is a residual effect rather than a continuance of the disease. In secondary cases the duration is more uncertain. When left alone a serous effusion generally clears up, but it may be long before the breath sounds and percussion note become normal. This is due to the slow absorption of fibrinous exudation, adhesions interfering with full expansion, and partial collapse of the lung. A tuberculous effusion may be very chronic, quite temporary, or become infected with organisms causing empyema. Rarely it is purulent from the onset, perhaps due to rupture or extension from a caseous gland or a caseous focus near the surface of the lung. Such a case occurred in a girl aged 4 years. The dulness began over the manubrium and gradually extended to the right. Thick caseous pus was eventually evacuated through the axilla and the child slowly recovered.

Sudden death may occur from cardiac inhibition in large effusions, especially when there is much displacement of the mediastinum. It may be caused by sudden changes in intra-pleural pressure or vascular intra-thoracic conditions; the result of cough, movement, sudden change of position, rapid removal of fluid and irrigation. Occasionally it is due to pulmonary embolism or thrombosis, myocardial degeneration or rupture of an empyema.

A small purulent effusion can be re-absorbed, the fluid parts being first absorbed and then the solid parts undergoing fatty degeneration and necrosis; or a caseous mass may be left behind, a potent source of future mischief. Ordinarily it goes on increasing in size and eventually ruptures, leaving a permanent sinus and perhaps finally ending in death from amyloid disease.

Thus the prognosis of serous effusions is good, unless the lung is permanently bound down by adhesions, a rare event in children. The duration is not a certain guide for in some long standing cases expansion occurs at once, while in others of much shorter duration permanent collapse may be present. In tuberculous disease the prognosis is that of tuberculosis. The prognosis of empyema is bad in the acute primary cases. In the other forms it depends on the age of the patient, the complications and causation of effusion, early diagnosis, and to a slight extent on the methods of treatment. In the first and second year the mortality is very high, but few cases die subsequently to this age. Speaking generally, the percentage mortality of children under 5 years of age is about the same in each year of life in cases treated by incision, and is rather higher in the first two years in cases treated by resection. The prognosis is particularly bad in the first year. The causes of death have apparently little to do with the nature of the operation and include bronchitis, pericarditis, broncho-pneumonia, œdema of the lungs, general tuberculosis, purulent meningitis, and



occasionally syncope during irrigation or erysipelas starting from the wound. There is a certain amount of evidence that the prognosis is worse in mixed than in pure infections, and in streptococcal than in pneumococcal cases, unless there is a polyserositis or a general pneumococcal blood infection. It is especially bad in bilateral cases and those secondary to tuberculosis. It is better, if the organisms stain feebly and grow badly (Bythell). If they are of high virility, the prognosis varies as the degree of leucocytosis. Deficient leucocytosis is a bad sign. If the empyema is left alone, death may result from pressure on the heart, acute toxæmia or some complication. The patient may recover by absorption of the pus or rupture of the abscess and discharge through the lung or chest wall. In such an event the illness is prolonged, its course uncertain and the risks grave. Even if recovery does result, there is usually a deformed chest, shrunk side, approximated ribs, depressed shoulder, curvature of the spine, diminished movement of the chest, displaced heart, fibroid change in the lung, and secondary tuberculosis or lardaceous disease. The lung is small, solid, carnified, retracted and covered by a dense layer of thickened pleura. Similar results may follow approved methods of treatment in old standing cases. In some of these re-expansion of lung is complete. Untreated cases commonly die from asthenia, amyloid disease due to chronic discharge, or some complication. In purulent pleurisy, differing from empyema merely in degree, absorption does take place and may leave no ill effects. The lung re-expands more readily in children than in adults, but there is greater liability to complications.

*Treatment.*—Acute serous effusion is treated during the febrile stage on the same principles as dry pleurisy. It may be necessary to evacuate a large and rapidly increasing effusion. Generally the child is kept in bed until the fluid is re-absorbed; and is not allowed to sit up or make any exertion if there is much effusion. Absorption is apt to be slow, and possibly can be hastened by counter-irritants and mercurial applications. Thiosinamin and fibrolysin injections have been recommended. Special care is necessary during convalescence because of the possibility of tuberculosis. Breathing exercises encourage the expansion of the lung.

A serous effusion must be evacuated if dyspnœa is excessive; if there is mischief in the opposite lung, indicated by wheezing, rales, and bronchitic or blood-stained expectoration; if there are attacks of faintness or syncope; if the patient is restless, unable to take food properly and unable to sleep, or develops nausea, vomiting or diarrhœa; if the fluid has existed for more than 3 weeks and is not commencing to subside. Early tapping is only necessary if there is serious dyspnœa from rapid and excessive effusion. It neither reduces the fever nor shortens the duration of inflammatory process. The fluid very soon re-accumulates and often to as great an extent. It may remain stationary for a long time and then clear up in the course of a few days. I have never come across a case of persistent effusion in a child.



untreated by paracentesis, so this operation is probably rarely necessary except with the object of hastening recovery.

The risks of *paracentesis* or tapping are those of exploration, *plus* certain dangers due to the particular method adopted. Syphonage is much safer than aspiration. The apparatus consists of a small trocar and cannula, 2-3 ins. long, 3 feet of rubber tubing with a short piece of glass tubing interrupting its continuity about 4 ins. from the end, a test tube, bull-dog forceps, thread and a large beaker or basin. The tubing is tied on to the cannula with thread and the trocar is pushed through the stretched rubber into the cannula. The other end of the tubing is tied so that it rests in the test tube, reaching the bottom. Both tube and test tube are filled with sterile water or salt solution and clamps put on. The trocar and cannula are plunged into the chest and the trocar removed, the clamps are taken off and syphon action is at once set up. With the patient on the back in bed and the receiver on the floor a suction action is exerted equal to that of a column of water about 2 feet high, and it can be at once stopped by raising the test tube to a level with the hole in the chest. This pressure is sufficient to remove all fluid which can be taken away with safety. As the fluid flows out the lung re-expands and the heart slowly returns to its normal position. Cough, perhaps paroxysmal, may be induced by irritation of the lung by the cannula, or depend on expansion of the lung. Occasionally dyspnoea, pain, faintness and general discomfort ensue. If they are severe, they may necessitate temporary or complete cessation of the operation. Withdraw the cannula if the fluid becomes blood-stained. The glass tube enables one at once to note any change in the character of the fluid.

An aspiration bottle can be used by exhausting the air slowly and with extreme care after the cannula has been inserted into the chest, stopping at once if cough, pain or discomfort is induced. The rapid removal of fluid may cause sudden and fatal syncope; rapid transudation of a serous fluid into the lungs, leading to an alarming profuse expectoration and signs of suffocation; or pneumothorax and subsequent pyo-pneumothorax from rupture of a lung bound by adhesions or unable to expand. Treatment by incision and drainage is justifiable in a case which does not clear up after repeated tapping.

*Empyema*.—As soon as the presence of pus in the pleural cavity has been diagnosed measures should be taken for its free evacuation. Although cases have got well if left alone, and after tapping, the prospect of cure is so small and uncertain and the risks are so great that free drainage should be adopted. Paracentesis is useful to reduce the size of a large effusion and ward off temporarily acute distress, and for an empyema on one side while that on the other is drained. Babies are liable to collapse if a large effusion is evacuated rapidly. Gentle aspiration is preferable to syphonage, for the pus may be thick. Both methods are unsatisfactory and incomplete.



The notion that a successful result is based on the filling up of the cavity and sinus, by granulation tissue and adhesion of the visceral and parietal pleura, leads to bad methods of treatment. Such a course is extremely slow and full expansion of the lung can only take place subsequently. Cure is effected by expansion of the lung, elevation of the diaphragm, and falling in of the chest walls. If the lung expands quickly, adhesions form and the sinus rapidly closes. The cavity is drained by simple incision, or resection of a portion of one or more ribs. The presence of pus is first located by exploration and the incision is made with the exploring needle in position. Without this precaution the operation may be done in the absence of pus or a localised empyema may be missed. Valvular drainage, intermittent drainage, drainage by continuous, negative, hydrostatic pressure, and the removal of fluid and injection of air have all been tried.

It is claimed for resection that better drainage is secured; that masses of fibrin can be more easily removed; that the boundaries of the cavity can be explored and all loose adhesions broken down, not an unmixed advantage for it may convert a local empyema into a general one; that there is less danger of hæmorrhage, and that recovery is more rapid. It is a more serious operation; the medullary cavity of the rib is exposed and forms an absorbing surface; and the rapid growth of callus soon diminishes the orifice.

The advocates of simple incision point out that the operation is simpler and more quickly performed, that the shock and the anæsthetic danger are less, and that the risk of pyæmia is smaller; that a large oval opening is easily made and drainage is quite efficient. These arguments are particularly applicable for babies under 2 years. The risk of hæmorrhage is infinitesimal, if it is remembered that the artery runs along the under border of the rib. The assertion that resection is usually a quicker and more certain cure than simple incision is not borne out by clinical experience. Post-mortem evidence proves that drainage is quite efficient in the simpler operation. If at the time of operation the ribs are too close together to get in a good sized tube, two smaller ones can be put in side by side. Next day it will be found that the ribs are more widely separated and a larger tube can be inserted. Resection should be reserved for those cases, rare in children, in which the ribs are so close together that good drainage cannot be secured without; for those in which a high temperature and offensive discharge, subsequent to the simpler operation, indicate insufficient drainage; for those in which partial re-expansion of the lung and falling-in of the chest wall have been insufficient to close the sinus. In the primary operation an inch or more of the rib is removed and the periosteum left. For the closure of a sinus it may be necessary to remove portions of several ribs or Estländer's operation, a dangerous and unsatisfactory mode of procedure, may be requisite. If not fatal, it results in fearful deformity.



General anæsthesia is preferable to local, when exploratory punctures are necessary as a preliminary. Chloroform or ethyl chloride is used. Light anæsthesia is sufficient and may be stopped as soon as the pleura is opened. The child will then cough and assist expulsion of fluid. The anæsthetic is given with the patient on the back near the side of the operating table and the operation completed without altering the child's position. Alarming or fatal collapse may result from turning the patient over on to the unaffected side. Local anæsthesia is advisable in large effusions on the left side and in bilateral cases.

The incision should be made in the fifth or sixth space in the mid-axillary line or just behind it. This is the most satisfactory place for drainage of a general empyema, for the comfort of the patient and the convenience of the surgeon. If the opening is made behind, the patient is apt to lie on the unaffected side and the action of gravity is opposed to effective drainage. If the opening is made lower down, the diaphragm may ascend and partially close it; or the diaphragm may be injured should the opening be lower than the seventh space on the right side and the eighth on the left. The diaphragm rises higher in the chest than in adults. In a child on its back the most dependent part of the pleural cavity is in the posterior axillary line or just anterior to it.

The incision is made in the middle of a space,  $1\frac{1}{2}$ -2 ins. long, and the hole is kept open with dilators to allow masses of fibrin to escape. Do not be anxious to empty the cavity. If the patient shows any bad sign, insert a tube and put the dressings on quickly. Evacuate large effusions slowly. Do not use a long tube, one about 2 ins. long and of as large a calibre as convenient is big enough. The origin of the use of a long tube is the erroneous belief that the cavity heals by granulation from the bottom. Holes in the side of the tube are of little value for assisting drainage, which takes place chiefly by the sides, and the tube is merely a temporary expedient for maintaining the opening. Use a flanged rubber tube or a straight one transfixed with a large safety pin to which tapes are tied to go round the chest. Put antiseptic gauze under and over the safety pin, cover with a large pad of cotton wool and dress twice daily at first. At each dressing the tube is removed and washed. A new tube may cause increased discharge, perhaps because of the sulphur in its substance.

The removal of the tube depends upon the state of expansion of the lung, the duration and the size of the empyema, the character of the pus and the organisms present, and the condition of the patient. It is usually left in too long and acts as a foreign body creating a permanent sinus, maintaining the discharge, and possibly causing necrosis of a rib. It is generally removed in 2 or 3 weeks but in many cases it can be left out in a few days, if the discharge is scanty and serous. The wound closes in another few days and a rapid and perfect cure ensues. The chief risk of early removal is the accumulation of a small amount of pus or sero-pus beneath the scar. Such a collection is treated as a simple abscess.



Occasionally the use of a short tube, or even a long one, allows a pocket of pus to be shut off by adhesion, keeping up the temperature and malaise. It can generally be found on exploration with a probe. After either incision or resection the sinus heals in 3-7 weeks, sometimes more and occasionally less rapidly.

Exploration with the finger is of doubtful value. There is no advantage in scraping away thick adherent lymph. Only detachable flakes and masses, which would come away in the course of a day or two, can be removed with safety by this method. Scraping may produce raw bleeding surfaces for the absorption of septic products. Irrigation is necessary in putrid effusions but the utmost care must be taken to permit the free escape of the fluid. It may cause fatal syncope, said by some writers to be due to the use of irritant injections. It is best to use saline lotions or the iodine bath.

The after-treatment consists of frequent dressings and antiseptics if there is much discharge. The tube is removed and cleaned, the child is turned on the side and drainage assisted by judicious compression of the chest. Drainage may not be perfect after either incision or resection. Sometimes a second or so-called counter-opening is needed.

In order to induce re-expansion of the lung after the sinus is closed the child can be taught to blow soap bubbles, trumpets, or fluids from one bottle to another, breathing exercises and the use of dumb-bells. These methods are applicable with the sinus still open and can be assisted by an apparatus to create negative pressure in the pleura. There is no immediate necessity for this treatment as extraordinary improvement occurs in time. Whooping cough is sometimes markedly beneficial.

The causes of failure after operation are too small an opening, inefficient drainage and accumulation of pus; thick pleural adhesions, defective breathing and weak respiratory muscles; and complications. A persistent sinus is due to imperfect drainage, non-expansion of the lung, dead bone, tuberculous pleurisy or caries of the spine. Occasionally adjacent ribs become united by bony bridges and help to keep up the fistula. Bier's suction cups applied several times daily are useful for encouraging contraction of the sinus.

A localised empyema is treated on similar principles. If an empyema is due to tuberculosis, operate unless the condition was hopeless before the empyema formed. Remove the fluid by gentle aspiration. If the lung seems not much diseased and able to expand, incise and drain. In chronic cases in which the lung has little power of expansion and the effusion is only producing symptoms by mechanical interference with the heart and lungs, an occasional partial aspiration is sufficient. Should the case go on well an incision should be made in the hope that the chest wall will fall in and the cavity granulate up.



*Bilateral Empyemata* are treated on similar lines. If there is general effusion on both sides, remove some of the fluid by aspiration to relieve the heart and lungs. On the next day drain the left side, and operate on the right side a few days to a week later. If the effusion is general on one side and localised on the other, drain the general effusion first. Bilateral localised effusions can be drained simultaneously.

The successful treatment of empyema depends on early diagnosis, early and efficient drainage, removal of the tube as soon as the discharge becomes scanty and serous, and the absence of the various complications on which fatal results usually depend. Many cases go wrong through mismanagement. It is advisable that the treatment of these cases should be left entirely in the hands of the physician, at any rate after the drainage tube has been first inserted.

**Pneumothorax.**—Pneumothorax is rare and almost invariably due to tuberculous disease of the lung, injury or rupture of an empyema; rarely to emphysema, pulmonary abscess, gangrene or hydatid. The symptoms are the same as in adults. An acute effusion of air is ushered in with pain, alarming dyspnoea, cyanosis, rapid fall of temperature, and all the signs of severe collapse. More often there is a slowly increasing shortness of breath. The chest is in a position of inspiration, immobile and hyper-resonant, and the heart is much displaced. The opening varies in size and shape, and may be multiple in caseous phthisis. If there is fluid, splashing, metallic tinkling and bell sound may be obtained. The breathing is amphoric if the opening is large and tinkling may be heard, due to fluid dropping from the aperture into the cavity on coughing.

It may be necessary to relieve urgent symptoms at the onset by syphonage. Later on, tapping is required to get rid of the fluid which is poured out. Sometimes the air is absorbed and then the fluid, but often it is necessary to evacuate the fluid by syphonage if it is serous, and by incision and drainage if it is purulent. In syphonage it is important to keep the mouth of the cannula below the level of the fluid; it will not create enough pressure to cause the weak place in the lung to give way. The prognosis is bad in hydro-pneumothorax due to tuberculosis and still worse in pyo-pneumothorax.



## CHAPTER XL.

### THE HEART AND CIRCULATION.

*The Heart at Birth—Examination of the Heart—The Pulse—Functional Heart Murmurs—Functional Disorders—Diseases of Blood Vessels—Œdema and allied conditions.*

At birth the heart weighs from  $\frac{1}{2}$ -1 oz. ; varying with the bulk of the body, though relatively greater than in adults. It is equal to one-seventh that of the liver, and doubles its weight in about 2 years. Up to this time its ratio to the body weight decreases and after that it increases. At puberty there is considerable increase in size. The walls of the ventricles are at birth almost equal in thickness but at the end of the second year the left ventricle is double the thickness of the right. It is situated more to the left than to the right, rather more horizontally and higher up than in the adult, and the apex is consequently higher up and further to the left. The apex is the lowest and most external point at which the cardiac impulse can be felt. It varies greatly with the position of the child and should be noted in the dorsal decubitus at the beginning of any illness. Up to the end of the third year the apex may be as much as  $\frac{1}{3}$  in. outside the nipple line in the fourth space. It is then felt in or near the nipple line, and after the seventh year is from  $\frac{1}{4}$ - $\frac{1}{2}$  in. within the nipple line in the fifth space ; before that it may be either in the fourth or the fifth. The position is modified by deformities, cardiac, pulmonary and pleural affections, and other causes. Pericardial fat is absent at birth and remains scanty during childhood. A murmur may be present during the first few days, due to an open ductus arteriosus.

**Examination of the Heart.**—In every patient the heart should be examined with the chest bare, for heart affections are common in early life and their onset is often overlooked. Examination is easy since it is rarely interfered with by emphysema, adiposity, great muscular development or pendulous breasts. Be careful that the hands are warm.

First localise the apex. Dilatation of the right ventricle pushes the apex upward and outward, along the arc of a circle with its centre at the aortic valve and a radius from this point to the apex. The aortic valves are the most fixed point and are situated under the middle of the left third chondro-sternal juncture. In dilatation of both ventricles the apex is displaced outward only, and in dilatation of the left ventricle downward.



Note the character of the impulse and any precordial prominence. Pay special attention to evidence of dilatation or hypertrophy. The heart of the child is precocious in its development and relatively hypertrophied, especially in active boys. Unless there is evidence of dilatation, such as increased dulness along the left border of the heart and epigastric pulsation, this condition is unimportant. Palpate gently and percuss lightly, without using a pleximeter. Pulsation and dulness to the right of the sternum are important facts. Dulness in the fourth right intercostal space is normal and due to the auricle. If it is increased, it may extend  $\frac{1}{2}$ -1 in. from the sternum and about half this distance in the third space. In great dilatation there may be even dulness in the second space. The presence of this dulness is often of grave importance and indicative of the necessity for leeches. The normal limit of cardiac dulness to the left is one finger-breadth inside the nipple line, sometimes it reaches the nipple line and occasionally extends a finger-breadth beyond it. After infancy, dulness beyond the nipple line is pathological.

For auscultation use a binaural stethoscope, unless thoroughly competent to use a wooden one without undue pressure on the chest. The pulse and general appearance of the patient may give more valuable evidence than auscultation. It is excellent practice to make a provisional diagnosis before using the stethoscope. By such a method the doctor gets into the habit of properly appreciating the effects of disease on the heart and not merely on the characters revealed by auscultation. The pulmonary second sound is increased in mitral stenosis, mitral regurgitation and generally in congenital morbus cordis. This is a striking feature for on account of low blood pressure the aortic second sound is feeble. If a murmur is present, note the time, quality, distribution and point of maximum intensity; so too in the case of thrills. Above all do not confuse facts with inferences.

**The Pulse** is not felt during the first quarter of a minute after birth. The heart then begins to beat slowly at the rate of 10-12 beats per minute. In the second half minute the child usually cries and the pulse rate goes up to 160. After that it falls to 130-140. Both the pulse and respiration rate are normally higher in infancy and early life than in adults. Both are increased in fever but the relative increase in the pulse rate is less than in the adult. In the infant for each rise of a degree in temperature the pulse rate increases 5 beats per minute; in early childhood 7; and in the adult 10. The infant's pulse would become dangerously rapid if it increased 10 beats with every rise of 1 degree in temperature. Anything over 160-180 beats per minute is bad. The respiration rate increases more than in adults rising 3 in infants, 2-2 $\frac{1}{2}$  in early childhood, and 2 in adults for each degree of temperature. The pulse rate is four times as frequent as the respiration rate, but with rise of temperature the ratio decreases down to 3-1 at 105° F. The pulse rate is roughly in the first year 100-130; second year 110-125; at 2-5 years 100-115; at 5-10 about 100, and up to 13 years, 80-90. The



*blood pressure* is low for the lumen of the vessels is relatively greater. The pressure in infancy is 80-85 ; up to 7 years 85-95 ; up to 10 years 95-100 ; and up to 13 years 100-110. All these figures may be modified by disease of the heart, lungs or brain, and typhoid fever, etc.

An intermittent pulse is frequent during sleep and under chloroform. Irregularity is present in nervousness, tuberculous meningitis, organic heart disease, gastro-intestinal troubles, appendicitis, anæmia, chorea, during convalescence, and from the effects of toxins and drugs. Irregularity in rate or rhythm is much less important than in adults. Increased *pulse rate* occurs in fevers, toxæmia, pericarditis and effusion, and nervousness. In endocarditis the pulse is frequent, strong, full and often irregular. In myocarditis increased frequency is associated with gallop-rhythm if the affection is acute, and with irregularity if it is chronic. In hypertrophy both rate and volume are increased unless there is aortic stenosis.

The rate may be so frequent as to deserve the name of tachycardia. A very frequent pulse is sometimes present in children taking Tibble's Cocoa and is associated with nervous irritability. *Bradycardia* or *Oligocardia* is usually unimportant unless it is associated with irregularity. It may be due to disorders of the central nervous, digestive, respiratory or genito-urinary system ; or to the poisons of lead, alcohol, tobacco or digitalis. It occurs in diphtheria, cardiac disease, anæmia, asthenia and certain skin affections. It is not infrequent in convalescence from acute fever and is common at the onset of tuberculous meningitis. Sometimes it has occurred paroxysmally.

**Functional Heart Murmurs.**—Most writers state that functional murmurs are rare under 4 years of age. This is certainly not true of the systolic murmur which is heard over the pulmonary area and is quite common. It is a functional, inorganic or hæmic murmur, with its maximum intensity over the second left interspace close to the sternum, sometimes more to the left or over the third left cartilage. It may be heard only in the second left interspace ; over an area from the second to the fourth spaces ; just above and within the apex ; or over the sternum, at the apex and even beyond it. Occasionally it is heard over the aortic area or is associated with a similar murmur in this situation, due to the same cause, or with a venous bruit in the neck and over the manubrium, and a systolic murmur over the carotids and subclavians. The typical systolic pulmonary murmur is superadded to the first sound, which is distinctly audible ; sometimes there is a distinct interval between them. It varies in duration and character from soft and blowing to musical. It is increased in loudness in the recumbent posture and perhaps only heard then. It is increased by exercise, may disappear on deep inspiration, and is best heard at the end of forcible expiration. The second sound is usually accentuated. The cardiac dulness is not increased and the position of the apex is unaltered, unless there is anæmia and cardiac dilatation.



This murmur may be due to anæmia, but is not necessarily so caused. It may be absent in severe anæmia, and loud although anæmia is slight or absent. It is probably dependent on the low arterial tension in the pulmonary artery and a dilatation of the artery causing a relative stenosis at the orifice. Arterial tension is normally lower in children than in adults and is more easily reduced. The lumen of the main vessels is wide and the cardiac cavities are small. Both the pulmonary artery and the aorta are more easily dilated than in adults, but the murmur is more readily produced at the pulmonary orifice because of the thin wall of that artery. The murmur must not be confused with that due to anæmia from which it cannot be diagnosed with certainty, except by a blood count and hæmoglobin estimation. It has also to be diagnosed from the organic murmurs of pulmonary stenosis and mitral regurgitation, the pressure of enlarged bronchial glands, and possibly the pressure of a dilated aorta on the pulmonary artery.

Diastolic functional murmurs are very rare. A cardio-pulmonary one is less common than in adults. It is a short, whiff-like or squeaking, systolic murmur due to the impact of the heart on the lung or compression of the lung by the heart. It is heard best at the apex and along the left border of the heart, and may be transmitted a little to the left. It is loudest at the end of inspiration and may disappear on expiration, especially in recumbency.

**Functional Disorders of the Heart** are rare before the seventh year but are not infrequent about puberty. The most common causes are indigestion and nervous exhaustion. Sometimes there is a definite exciting cause such as fright, tea, coffee, cocoa containing kola nut, or cigarette smoking. Sometimes it occurs during or after acute febrile disease, more especially those in which myocardial degeneration is apt to develop. At puberty the rapid growth of the body may exceed the development of the heart and is a frequent cause of undue rapidity of the heart's action, particularly if it is associated with anæmia, overpressure at school or over-exertion.

The chief symptoms are palpitations, tachycardia and occasionally bradycardia. A slow heart has already been referred to under the pulse. Paroxysmal attacks of palpitation, lasting for a few seconds to hours at a time, produce the same symptoms as in adults.

*Tachycardia* is the most interesting of these affections. The attack comes on at varying intervals and is of variable duration. It is usually induced by exertion or fright, and comes on quite suddenly. The symptoms include pallor, anxiety, sweating, exhaustion and palpitations. The breathing is not accelerated and the urine is increased in quantity. Sometimes the attacks are associated with repeated vomiting, though they persist after the vomiting has ceased, or with constipation. The pulse may be uncountable and almost imperceptible. Tachycardia is probably a true neurosis and independent of any heart lesion. It has been ascribed on



insufficient evidence to vagus palsy, compression of the vagus by enlarged glands, accelerator stimulation or bulbar mischief. It has followed pertussis and measles.

The most useful treatment is by bromides and valerianates, and careful regulation of the digestion, bowels and mode of life. Stimulating foods and drinks must be avoided. During the attack a cold compress, ice bag, spray of ether or ethyl chloride, or mustard leaf may prove beneficial.

*Syncope.*—Many diseases end in syncope, sudden and permanent cessation of the heart's action. "Fainting" or "Swooning" is a mild form of syncope, more gradual in development and of temporary duration. The loss of consciousness is due to the failure of cerebral circulation, or alteration in intracranial pressure. An actual fall in blood pressure is the chief determining factor. This is brought about by inhibition of the heart and vasomotor centre, and vaso-dilatation. Sudden death in aortic regurgitation seems due to sudden diminution of intracranial pressure. In other cases the cardio-vascular derangement, diminishing the cerebral blood supply, is due to stimulation of the cardio-inhibitory centres in the medulla, e.g., by sudden intense pain, infantile colic, various sights and odours, and emotion. Syncopal attacks can be induced by sudden intense cerebral congestion, as in the paroxysms of pertussis.

Fainting is apt to occur in school-children during morning chapel. The erect posture, empty stomach, lack of cerebral stimulation and the ill-ventilated building are all exciting causes. These children often exhibit albuminuria, a sign of vasomotor instability, and in some no doubt the fainting attack is dependent on masturbation before rising. Fainting is most common in girls about puberty and is then a sign of hysteria. The chief causes are the sudden assumption or prolonged maintenance of the erect posture; rapid loss of fluid as in hæmorrhage, profuse purging, or the evacuation of ascitic fluid; the vaso-dilatation induced by heat after the superficial vessels have been constricted by exposure to cold; and the purely nervous causes already mentioned. Fainting is ushered in by pallor, a sense of increasing feebleness, dimness or loss of sight because of lack of blood supply to the retina, sighing respiration, and frequently undue consciousness of the heart's action and a sense of nausea. A sinking feeling in the epigastrium is due to splanchnic vaso-dilatation. Consciousness is lost, more or less completely, for a variable time but not so suddenly as in epilepsy. The child sinks down or can lie down, and rarely falls so suddenly as to cause injury. The heart beat may be very feeble and the radial pulse almost imperceptible. Consciousness and colour return gradually, without any mental confusion or special tendency to sleep.

A severe faint may be attended with muscular spasm. In minor epilepsy it may be absent. It is common for petit mal to be diagnosed as fainting. In minor epilepsy there is no initial facial pallor, loss of consciousness is sudden and recovery as sudden, and there is frequently some



mental confusion, hysteroid symptoms or sound sleep immediately after. Both are due to the failure of the cerebral circulation, but differ in the conditions of their production and tendency to recurrence. A family history of epilepsy may be present or an exciting cause of the fainting attack. Even babies have fainting attacks which are extremely difficult to diagnose. Swooning is treated by fresh air, recumbency, lowering the head, loosening the clothes, a cold douche, stimulants and smelling salts.

**Functional Circulatory Disorders** might strictly be included in the section on nervous diseases. The two chief ones are Chilblains and Raynaud's Disease.

A *Chilblain* (*erythema pernio*) or *Frost-bite* is a local vascular lesion dependent on altered conditions of the blood or circulation. Primary vaso-constriction is followed by vaso-dilatation and in this respect it is analogous to Raynaud's disease. The first effect is local anæmia, and it is quickly followed by hyperæmia due to venous engorgement and transudation, and then by thrombosis and even gangrene. Observations on the blood have shown, in one class of case, a deficient coagulability and consequent liability to serous exudation or "serous hæmorrhage," like that of urticaria. The predisposing factors are heredity, lowered vitality, feeble blood supply to the extremities, insufficient exercise and a deficient supply of fatty foods. Children, shop girls, old people and the feeble-minded are very liable; so, too, the anæmic, those with a tuberculous tendency or morbus cordis, and those who live a sedentary life. The main exciting cause is rapid change in external temperature.

In the early stage there is an erythema and secondary inflammation. The heel, toes, fingers, nose and ears are the common sites. The part affected is swollen, exhibits a superficial, dusky or bluish redness, with tenderness, itching and stiffness of the skin. The itching is worst when warm. In mild cases the skin desquamates and the chilblain gets well. If neglected, severe or irritated, vesicles form and it becomes "broken" or ulcerated. Cracks develop, are infected, and may lead to deep ulcers or more serious mischief. In still worse cases the damaged part becomes gangrenous, a line of demarcation forms, and it gradually separates and drops off. The diagnosis is quite simple. Mild Raynaud's disease may be overlooked. Recurrence is common.

To prevent chilblains attend to the general health. Give cod-liver oil for malnutrition; iron for anæmia; and drugs such as arsenic, strychnia, ergotin, belladonna and digitalis. Calcium salts are often useful, by increasing the coagulability of the blood, if given for only 1-3 days at a time. Ichthyol pills, after food, can be tried. Maintain vascular tone by alternate hot and cold douching of the limbs for 15 minutes nightly. Guard against cold, damp feet and hands, and order plenty of exercise. Rub the extremities with sp. camph. every morning, camphorated vaseline. gr. 75 ad oz. 1, or camphorated soap. In the erythematous itching stage apply Bier's



method of artificial hyperæmia ; commercial formalin, half strength, once or twice daily, and then vaseline or boroglyceride ; tr. iodi, iodine 2 per cent. in collodion, silver nitrate solution or sp. camph. ; rubbing with methylated spirit or whisky and water ; liniments of aconite, belladonna, chloroform and soap ; bals. Peru, ichthyol or thigenol, 10 per cent. in oil or lanolin ; or with tannin gr.  $\frac{1}{2}$  ad oz. 1 glycerine and rose water, p.a. Bœck of Christiania recommends ichthyol, resorcin, tannin  $\overline{aa}$  gm. 1, aqua 5 c.c., painted on nightly, it forms a varnish in a few minutes and causes the œdematous swelling to disappear, the part looking black for 1-2 weeks. If there is much inflammation apply ung. plumbi oz. 1, vaseline oz. 1, ol. olivæ oz.  $\frac{1}{2}$ , ac. carbol. gr. 10-20, ol. lavandulæ m. 20, freely on lint at night.

Open blisters and dress them with ac. carbol. 2 per cent. in Carron oil, or with borated vaseline. Broken chilblains can be dressed with walnut leaves soaked in hot water, or lint steeped in hydrag. perchlor. 1 in 2000 ; painted with tr. benzoin. co. ; powdered with bismuth. salicyl. 1, amyli 9 parts ;  $H_2O_2$  (10 vol. strength), mixed with an equal amount of hot water and applied for 15 minutes twice a day ; weak ointments of Friar's balsam, liq. carbonis detergens or ol. rusci ; ung. zinci ; hydrarg. ammon. gr. 5, ichthyol gr. 10, zn. oxid. dr. 2, amyli dr. 2, vaseline oz.  $\frac{1}{2}$ . Lewis Jones recommends an electric footbath for 10-15 minutes nightly. The main principle in the treatment of broken chilblains is that of all open sores. Protect them from infection, keep them clean, and apply soothing or stimulating ointments according to the stage of the ulceration. If the ulcers do not heal touch them with solid nitrate of silver.

*Raynaud's Disease* is a neurosis, a vasomotor disturbance, a disorder of function and not of structure. It affects the fingers, toes, ears and nose ; less often the hands, feet, face, tongue and trunk. Children suffer less severely than adults. There is less functional impairment, with slight ulceration and destruction of tissue and practically no general symptoms. Comby (1905) reported 3 cases, aged 9 and 10 years, with local asphyxia and dry gangrene of the ears and ends of the extremities.

The first stage is that of "Local Syncope" or *dead fingers*, due to excessive vaso-constriction. Pain may be considerable or absent. This condition may arise simply from cold and is not necessarily due to Raynaud's disease. The second stage, "Local Asphyxia," is usually but not always preceded by local syncope. It is produced by extreme vaso-dilatation and great venosity of the stagnant blood, giving rise to a purplish or black discoloration. It is very painful, especially when passing off. In the third stage there is dry "Gangrene" of the extremity or a local necrosis with the formation of sores, as in broken down chilblains and frost-bite. These necrotic areas heal readily in warm weather, leaving scars. Thus all the effects are such as can be produced by cold, and the main characteristic of the disease is that the subject is one who is unusually susceptible to cold. Hæmoglobinuria may occur at the onset and not



subsequently, during many attacks, or even independently. Purpura is less common and may be recurrent. Scleroderma and melanoderma of the face, trunk and joints have been reported. Cerebral symptoms are such as can be explained by vaso-constriction, viz., transient vacancy or loss of consciousness as in *petit mal*, temporary aphasia or disorder of speech, hemiplegia, delirium and mania; mental torpor; convulsive attacks of an epileptiform or hysterical nature. Impaired vision is due to constriction of the central retinal arteries. An ultimate cure may be effected. The disease is not fatal.

**Treatment.**—Avoid cold, fatigue and excitement. Wash in warm water, wear warm clothing, protect the hands, live in warm rooms and a warm climate. Treat local syncope in the same way as frost-bite with warm cotton wool, immersion in tepid water, and gentle active and passive movements. Vaso-dilators are useless. Gangrene is treated on ordinary surgical lines. Between the attacks attend to the alimentary system. Naphthol, sulphocarbolates and other intestinal disinfectants may do good. Electricity should be applied in the form of faradism to the spine over the site of origin of the affected vasomotor nerves; and to the affected limbs by immersion in a salt water bath through which a constant current, which can be frequently interrupted, is passed. A floating kidney may be fixed. Cushing recommends the temporary constriction of the affected limb for some minutes. On relaxation, the blood flow may overcome the spasm.

**Gangrene** is occasionally seen in the newborn as the result of pressure, asphyxia or of uncertain origin. It may follow infective fevers and is then generally due to thrombosis, secondary to endarteritis. At any age it can occur from microbial infection. Sometimes it follows the application of carbolic acid dressings, independently of tight bandaging, waterproof dressing or the nature of the primary injury. This is probably the effect of idiosyncrasy and is independent of age, sex, and the state of the health. Harrington (1900) collected 132 cases, some in children. It is a dry gangrene. The skin becomes dry, wrinkled and greyish white; then darker and more shrivelled, and a line of demarcation forms. Amputation is often needed.

**Diseases of Blood Vessels.**—Arteritis occurs in acute infections such as scarlet fever, typhoid fever, diphtheria, pneumonia and sepsis, and in congenital syphilis and rheumatism. Henning (1890) reported endocarditis and aortitis of a few weeks duration in an 8-months child who died shortly after birth. One valve of the aorta was contracted. The mother had rheumatism during pregnancy. According to Barrie (1905) acute rheumatic arteritis usually causes aortitis and may affect the coronary, peripheral and visceral arteries. It comes on at the end of rheumatic fever. Pain and tenderness are present in the course of the artery, the limb is swollen and cold, and there is no fever. It may clear up in 1-2 weeks, become chronic and cause arterio-sclerosis, or obliterate the artery. No case of secondary



gangrene is on record. If it affects the coronary artery it may cause myocardial changes, dilatation and sudden death.

Weisel (1906) found arteritis in 80 young subjects, many infants, secondary to diphtheria in 20, scarlet fever 20, and other infections 40. Similar changes were present in all. In 6-10 days minute yellowish patches appear in the aorta, carotids and coronary arteries; with secondary minute calcareous deposits in a few. It usually ends in regeneration, but occasionally in atheroma and arterio-sclerosis with their sequels.

Congenital syphilis is apt to produce aortitis and aortic atheroma, which may be associated with valvular lesions or with generalised arterio-sclerosis. Its signs are increased aortic dulness, murmur and thrill. Arterio-sclerosis often includes many varieties of endarteritis and arteritis; some congenital, others due to the various causes above mentioned. The term should be restricted to the type seen in chronic interstitial nephritis. Calcareous degeneration is a sequel of one or other of these affections. Calcification of all the peripheral arteries, the iliacs like pipe-stems and the aorta hardly affected, has been reported at the age of 6 months (Hale White); calcareous degeneration of the media of the aorta and pulmonary artery at 6 days (Durant); calcification round the mitral orifice at 2 months (Goodhart); calcification of the aorta and valves at 2 years (Moutard), and of the temporal arteries at 15 months (Hodgson); and a few cases in older children, all apparently non-syphilitic. Endarteritis and coronary sclerosis have both been found in infants aged 5 months. All these affections are rarely recognised in the young but it is by no means improbable that arteritis is fairly frequent in infective disorders and accounts for some of the rare cases of aneurysm, thrombosis and gangrene.

*Aneurysm* is a rare affection in children. One child showed aneurysmal dilatation of the pulmonary artery as the result of congenital heart disease. In a girl, aged 16, cerebral hæmorrhage was due to the rupture of an aneurysm of the left posterior cerebral artery, the result of infective endocarditis. In a boy, aged 18, aneurysm of the posterior tibial artery was caused by embolism from valvular heart disease. It was cured by ligature of the popliteal artery. Phänomenow (1881) found endarteritis and aneurysm of the abdominal aorta in a 7-months foetus; and Martin reported one of the ductus arteriosus at the age of 1 month. Including my two cases and excluding Phänomenow's and those of congenital origin, I have collected 38 under 20 years of age; 25 of them under 13 years old. The ratio of boys to girls is 2-1. In 31 the site was:—thoracic aorta 6, abdominal aorta 3, iliac 1, femoral 5, popliteal 2, tibial 4, carotid 3, axillary 2, and innominate, coronary, ulnar, dorsalis pedis and cerebral 1 each. No doubt many more cerebral cases are on record and are described under the head of the primary disease. The main cause is embolism, from simple or infective endocarditis; and others have been due to endarteritis, atheroma, and possibly injury. Several have been cured by ligature. Other cases have ended fatally from



the primary disease, cerebral hæmorrhage or embolism, rupture into the pericardium and into the trachea. The symptoms and treatment are the same as in adults.

**Diseases of Veins.**—Dilatation of the jugular veins and of the superficial veins on the chest is caused by pressure on the superior vena cava by enlarged glands, the fibroid contraction in mediastinitis or by tumours. Occasionally it is unilateral. Dilatation of the subcutaneous veins in the legs and elsewhere is uncommon. Hæmorrhoids are sometimes seen. Venous dystrophy of the cranial veins is a condition in which the veins of the scalp in infants are dilated and unusually visible through a thin scalp. It has been regarded by Fournier as a sign of congenital syphilis, but may be due to malnutrition and venous congestion from any cause, and is quite unimportant.

*Thrombosis* is a sequel of phlebitis, or dependent on altered conditions of the blood and circulation. It is found in the sinuses of the skull, the cerebral vessels, internal jugular (generally septic and secondary to ear disease), the superior and inferior vena cava, iliac and other veins. Thrombosis of the inferior cava is not very rare as the result of intra-abdominal tumours, but is quite exceptional from other causes. A striking case was under Dr. Gould May. A fat anæmic boy, 10 months old, had an attack of diarrhoea and vomiting for 2 days, after which he seemed unduly prostrate. Two days later he was feverish and in 48 hours the legs began to swell. When seen on the eleventh day of the illness he was found fat, anæmic and not rachitic. The legs were greatly swollen and the abdominal veins prominent, forming a *Caput Medusæ* above the umbilicus. Two days later the temperature went up to 105° F. and there was a little twitching for which no cause could be found. The left leg was much larger than the right, and the veins on that side much dilated. Apparently there was thrombosis of the left iliac and only partial thrombosis of the right vein. Five weeks later the legs were quite normal. Thrombosis is apt to occur during the course of, or in convalescence from, infective diseases, more especially typhoid fever and diphtheria.

**Œdema** is a symptom. Common causes are backward pressure from cardiac failure or obstruction, renal mischief, general malnutrition and toxæmia. Occasionally it follows over-feeding after a period of starvation. In renal disease the nutrition of the vascular epithelium is impaired, the blood is hydræmic, and there is a deficient elimination of water and sodium chloride. This salt is almost entirely eliminated by the kidneys. The pathology is practically the same as that of ascites (p. 348). Transudation into the subcutaneous tissues must be regarded as mainly dependent on impaired nutrition and altered blood states. Thus it is quite common to see a moderate amount of œdema in all wasting diseases, notably marasmus, towards the end. But there are other œdematous conditions which are of



different causation and, though not strictly of purely circulatory origin, may be here described.

**Angioneurotic Œdema.**—*Syn.: Acute Circumscribed Œdema—Acute Idiopathic Œdema—Non-inflammatory Œdema—Giant Swelling—Periodic Swelling—Urticaria Tuberosa.*—This affection is characterised by extensive, circumscribed, non-inflammatory swellings of the skin and subcutaneous tissues, the mucosa and the submucosa. It affects the eyelids, part or the whole of the head, face or one limb, lips, tongue, pharynx and larynx, joints, external genitals, viscera and, indeed, any part of the body. It appears and disappears with remarkable rapidity, and is commonly associated with intestinal symptoms and low coagulability of the blood. It is most common in young adults. Cases have been recorded in the third and fourth months of life and at one year, and in children between 5 and 8 years. It may occur at any age, even in the first week. Males are twice as liable as females except in adult life. It has been ascribed to exposure, injury, dietetic and psychical causes. Undoubtedly there is a marked hereditary factor in many cases. Quincke referred to this connection and Dinkelacher, a pupil of his, in the same year (1882) recorded its occurrence in a matchmaker and his infant son, and Valentin (1885) reported that a second son had been affected since the first week of life. In another family 22 out of 39 members in 5 generations were affected (Osler, 1888); 5 members of a family in 2 generations (Apert and Delille, 1904).

The attacks are dependent on a neurotic individuality, and are often induced by acid fruits and wines, special articles of diet and other causes. Many cases are allied to urticaria and are merely an exaggerated form of it, being due to toxæmia. In others the hereditary tendency is the main feature. The two factors may be associated. Perhaps it is a pure vasomotor neurosis. There is an actual exudation of serum, and sometimes extravasation of blood. In this respect it is comparable to Henoch's purpura. In a woman, aged 29, in whom laparotomy was done for a severe abdominal attack, the intestines were found much congested, with a cylindrical enlargement of the ileum, 2 ins. long, near the ileo-cæcal valve, elastic and not pitting (Harrington, 1905).

The onset is sudden, perhaps preceded by indefinite malaise. The swelling appears in a few minutes to a few hours and disappears equally rapidly. It gives rise to no discomfort or merely such as would be due to the unusual distension; sometimes sensory disturbances, e.g., fulness, throbbing, burning, pricking, numbness. It is whitish in colour and does not itch or pit on pressure. Occasionally it is reddish and followed by discoloration like purpura, for which it may be mistaken. It affects any part, may even cause exophthalmos, and is rare on the scalp. Fever, if present, is moderate. The gastro-intestinal symptoms are epigastric pain, colic, nausea, profuse vomiting and diarrhœa, perhaps preceded by constipation. Suppression of urine, albuminuria and hæmoglobinuria have been noted. Glottic œdema produces severe asphyxia.



Cases have to be diagnosed from the urticarial swelling due to stings and bites, cellulitis and purpura. Severe intestinal symptoms suggest peritonitis, appendicitis, Henoch's purpura, and even intussusception in infancy.

The prognosis must be guarded because of the liability to death from œdema of the larynx or acute pulmonary œdema. Recurrence becomes more frequent with advancing age. The intervals between the attacks vary greatly in different individuals. Slight scaling of the epidermis may follow the attack and the skin be left a little thickened.

Treat the neurotic state and the alimentary tract. Œdema of the larynx is treated by ice, adrenalin, scarification, and tracheotomy if it does not yield to the simpler measures. General measures are rest in bed, cold douching, ice or fomentations, cooling lotions, atropine, strychnia, calcium salts, bromides and tonics.

**Trophœdema.**—*Syn.: Hereditary Œdema of the Lower Limbs—Chronic Congenital Elephantiasis.*—Milroy of Omaha (1892) described a solid œdema of one or both legs, congenital, except in one child in whom it began at 12 years, in 22 members out of 97 of one family in 6 generations. Its occurrence has been recorded in other families:—8 out of 36 (Nonne), 8 in 4 generations (Meige), 4 in 3 generations (Tobeison), 5 males and 8 females in 5 generations (Hope and French). This last family was peculiar in that some of its members had acute attacks with violent pain. Instances in brothers or in sisters, and isolated cases have also been recorded. Rolleston reported cases in a boy aged 13, and his sister aged 16 years, with a peculiar œdema of the legs, more marked after exercise and warm baths, but disappearing after rest in bed for some days. The mother had been affected since 10 years of age and 5 other children had escaped.

The œdema is generally congenital but may not develop for several years. It is unilateral or bilateral, affects the lower limbs wholly or partially, and is limited by the larger joints. It increases gradually when the patients are up and decreases on lying down. The skin is pale and cold, natural in colour if the limb is warm, blue and liable to chilblains in cold weather. Usually there is a history of lividity at birth. Pitting is induced by pressure and leaves a pallid area for some time on account of the weak circulation. Lividity is increased by gravity. Mental condition, development, sensation and reflexes are normal. Blood examinations are negative. No obstructive disease of the heart or circulation has been found. There is no evidence of deficiency of valves in the veins, nor of lymphatic obstruction. The absence of skin changes is against lymph stasis. Possibly it is the result of excessive transudation, due to some inherent defect in the blood vessels or blood. If so, it presents analogies with hæmophilia. The condition seen in children, especially about puberty, in which the hands and feet are swollen, cold, and more or less livid (*acro-cyanosis*), often associated with orthotic albuminuria, is unlikely to be mistaken for this affection,



though it suggests a similar vascular pathology. Angioneurotic œdema is of much more sudden and temporary character.

Occasionally one sees infants with a fairly solid œdema of the feet, dorsal and plantar, involving the toes and perhaps extending above the ankles. If the weather is cold, the feet are cold and livid. It may have been present from birth or vary from time to time, sometimes in accordance with the amount of urine passed. Probably some of these cases depend on renal inadequacy or actual disease, and this diagnosis may be supported by morning œdema of the eyelids or conjunctiva. Sometimes urinalysis only shows a few hyaline casts and renal cells. The ultimate result and the pathological anatomy of these cases require investigation. In the future it may be found that all these different varieties of œdema are more or less identical in their pathology, differing in acuteness and chronicity, depending on a vasomotor neurosis, a circulatory or hæmic defect, a lymphatic peculiarity, or combinations of two or more of these several factors.

Hereditary œdema is of importance from a prognostic point of view. It is harmless and persistent; but troublesome by reason of the clumsiness and weight of the limb, and because of the coldness and liability to chilblains.

Bandages afford some support but the œdema appears above them. Rest and elevation are temporary measures of relief. Vascular tone is improved by ergot, digitalis and galvanism. The limbs must be kept warm and massaged daily in an upward direction.



## CHAPTER XLI.

### CONGENITAL HEART DISEASE.

*Cardiac Development—Fœtal Circulation—Pathogenesis—Malpositions—Defects in the Septa—Arterial Defects—Valvular Lesions.*

**Development of the Heart.**—In its earliest stage the heart is formed by the coalescence of two symmetrical ventral tubes into a single cavity. This becomes bent on itself and divided into an auricle and ventricle conjoined by a short narrow segment called the auricular canal. Opening into the auricle is the sinus venosus, formed by the coalescence of the allantoic, hypoblastic and lateral somato-pleural veins which convey to the heart all the blood in the body. From the ventricle the blood passes to the bulbus arteriosus through a much constricted part, the *fretum Halleri*, thence to the aorta.

In the next stage the auricular canal is divided by the ultimate fusion of two small tubercles which grow from the anterior and posterior surfaces until they meet. The auriculo-ventricular valves develop within this canal. The ventricular septum begins in the fourth or fifth week and is completed about the eighth week. It grows upward from the apex and its plane alters in course of development. Were it not for this alteration in the plane, the central part of the septum would join the left and posterior side of the bulb and shut off the left ventricle from all arterial connection. The aorta and pulmonary artery would then arise from the right ventricle. In the base of the septum is the “*Membranous Space*.” This is somewhat lozenge-shaped, situated beneath the anterior and right posterior aortic cusps, and eventually closed by a thin membrane. Its origin is uncertain but probably it develops from the posterior wall of the bulbus, not from the septum, for it has been found present in the absence of the septum. By completion of the ventricular septum the *Cor Triloculare*, consisting of one auricle and two ventricles, is formed.

Failure in the development of the membrane closing the membranous space leaves a patent interventricular septum, or an *interventricular foramen*. In the human embryo the foramen is closed during the second month by the development of the muscular band characteristic of the mammalian right ventricle. It develops from the inner layer of the ventricular muscle, bends inward to reach the base of the aorta and fuses at its origin with the flat band on the infundibular septum. In systole the margin of the band falls



across the foramen and closes it. The band may be absent or fail to join the septal band, especially in cases of pulmonary stenosis.

The septum of the bulb is completed a few days before that of the ventricle. It begins as two small tubercles on the anterior and posterior walls of the bulb between the fourth and fifth branchial arches. The course of development is from above downwards and antero-posteriorly. The plane gradually alters by rotation towards the left until it is placed transversely, the left vessel or pulmonary artery being in front of the right or aorta.

The auricular septum is not fully formed until the fifth month. It develops by the formation of two separate septa which coalesce. The primary septum, "septum primum of Born," starts from the superior surface of the cavity, a little to the left of the median line, and grows downward to unite with the anterior and posterior tubercles of the auricular canal. At first the septum is sickle-shaped and a large aperture is left, next a complete septum is formed between the two auricles, and then the central portion becomes perforated by a number of minute holes. One of these enlarges and persists, while the others close up. A secondary septum develops close to the primary one. It arises from the median line and grows down from the superior surface of the cavity. It is incomplete but the aperture does not coincide with that in the primary septum. The two septa fuse together and leave an oblique opening, the *foramen ovale*. The valve of this orifice is derived from the primary septum.

The *right auricle* consists of the auricular canal, or primitive part of the cardiac tube, and the auricle proper. The latter is developed as a diverticulum from the auricular canal. It consists of all that part of the auricle in which the muscle is arranged as *musculi pectinati*, and includes the appendix. The effects of its systole must be taken into account for it is not merely a passive open chamber. At first sight it is not obvious why the auricle drives the blood into the ventricle and not backward through the orifices of the great veins, unprotected by valves, nor why the blood from the inferior vena cava should pass through the *foramen ovale*.

*Venous pulsation* may be visible in the internal jugular vein during health and is much more visible in cases of backward pressure. Two waves of pulsation may then be seen in the veins. The first is auricular systolic in time and due to the blood stream being dammed by the systole of the auricle. The second wave is visible towards the end of ventricular systole and merges, in the later stages of backward pressure, into and absorbs the auricular wave. In backward pressure there is actual regurgitation. The auricular wave would be due to regurgitation were it not for the presence of certain structures which close the orifices of the great veins. In fishes, amphibians and reptiles all the veins unite to form the sinus venosus, which is guarded at its orifice into the auricle by two strong valves to prevent regurgitation. The sinus and valves are present in the mammalian embryo



in the early months of foetal life. During the third to the fifth month the left valve almost entirely disappears. The upper part of the right valve disappears and the lower part forms the Eustachian and Thebesian valves, which help to guard the opening of the inferior vena cava and coronary sinus. The sinus venosus is absorbed into the right auricle and its original orifice becomes divided into three parts for the vena cava superior, vena cava inferior, and the coronary sinus. In the basal portion of the right valve a large bundle of muscle develops and forms the right *tænia terminalis*. This muscle is the main factor in closing the orifices of the cavæ during auricular systole. The opening of the vena cava superior is more strongly guarded than that of the vena cava inferior. Clinically we find venous pulsation in the tributaries of the former vein before it is seen in those of the latter. The closure of the caval orifices is not due to any sphincter muscle action, for no annular fibres surround the termination of the vena cava inferior, and those round the vena cava superior are few and weak. The disappearance of these valves and the modifications in their structure in mammals are distinctly advantageous. Instead of a rigid mechanism we have one which will allow regurgitation during engorgement of the auricle. In reptiles and amphibians there is an interventricular foramen, and the right ventricle can relieve itself by driving blood through the foramen into the left ventricle.

*Myogenic Theory.*—The heart muscle originates and maintains contraction independently of nerve stimulation. A small node of tissue, the “sino-auricular node,” has been found by Keith and Flack at the mouth of the vena cava inferior. It is supposed to represent a portion of the sinus venosus from which the heart contraction starts. Further remains have been found in the “auriculo-ventricular node” (*Knoten of Tawara*), in the right auricular wall near the mouth of the coronary sinus. From this the auriculo-ventricular bundle (*Bundle of His*), arises and passes over the auriculo-ventricular septum, dividing into branches to the right and left ventricle. “Nodal rhythm” is a simultaneous contraction of auricles and ventricles starting at the auriculo-ventricular node. It occurs in most cases of severe heart failure.

*The foramen ovale* is surrounded incompletely by the annulus ovalis, composed of two limbic muscular bands united at one end and having a shape and arrangement somewhat like that of the merry-thought of a duck. In foetal life about one-third of the blood passes through the lungs and the remainder through the foramen ovale. As the limbic muscle bands contract during auricular systole, they pull forward the vena cava inferior and direct the blood flow through the foramen. At birth the diaphragm descends and drags down the orifice of the vein. Hence the plane, in which the limbic bands act, becomes more vertical in direction. The course of the blood stream is altered and the contraction of the bands assists in closing the foramen. Closure is aided by the rise of pressure in the left auricle, due to



establishment of the pulmonary circulation, and the fall of pressure in the right auricle. The two septa are more closely opposed and become united by adhesion. Closure takes place a few weeks or months after birth, but is often incomplete and leaves a valvular orifice. This orifice is of no importance for it is kept closed during life by the higher pressure in the left auricle. Patency of the foramen is due to mal-development of the primary or secondary septum of the auricle.

The *left auricle* is a modified development of the auricular canal. The pulmonary veins unite and enter the auricular canal by a common trunk. As the lungs grow forward over the heart and the heart sinks into the chest, the orifices of the pulmonary vein become separated into right and left veins and later on into upper and lower divisions on each side. As the orifices separate, the muscle of the auricular canal grows over and fills up the intervals, thus producing the structure known as the vestibule. Ultimately the vestibule forms the largest part of the auricle. The muscle fibres of the vestibule and of the left *tænia terminalis* close the orifices of the pulmonary veins and prevent regurgitation. In mitral stenosis the *tænia* becomes greatly hypertrophied.

The *right ventricle* consists of a sinus and an infundibulum. The latter is called the “*conus arteriosus* or *pulmonalis*” and communicates with the pulmonary artery. In the turtle’s heart there are three ventricles. The right gives rise to the right aortic arch, the left to the left aortic arch, and the central one to the pulmonary artery. In the mammalian heart the development of the septum shuts off the part corresponding to the central ventricle of the turtle. An abnormal septum is rarely present in the right ventricle ; still more rarely in other cavities.

*Branchial vascular arches.*—The five pairs of primitive arches do not all co-exist at the same time. The permanent vessels are developed by the persistence of certain parts and obliteration of others. The primitive aortic bulb is divided into 2 vessels, the aorta and pulmonary artery. The fourth and fifth pairs of arches arise from this primitive stem, and the other pairs are branches of a secondary stem from the fourth arch on either side. The fifth arch on the left side persists as the ductus arteriosus and gives off the pulmonary artery as a branch. The fourth left arch persists as the aorta into the descending part of which, just below the common origin of the subclavian and vertebral arteries, the ductus arteriosus enters. Thus the pulmonary artery is a branch of the fifth arch and any interference with its development, causing stenosis or atresia, will maintain patency of the ductus. The three remaining arches on the left side unite and form the carotids, coming off by a common root, the common carotid. On the right side the fifth arch is obliterated. The fourth persists as the subclavian and comes off by a common root from the aorta forming the innominate artery. The carotids arise in the same way as on the left side, but their common root unites with that of the subclavian to form the innominate.



Both fourth arches may develop and form a double tube round the trachea, uniting to form a common aorta. The subclavian and vertebral arteries then arise from the fourth arch in a similar manner to that on the left side. The coronary arteries are occasionally imperfect in development.

*Abnormalities in veins.*—Both the superior and inferior vena cava may be duplicated. Both veins have been found entering the left auricle; the pulmonary vein opening into the right auricle; the right and left innominate veins opening separately into the right auricle; and the left vena azygos entering the coronary sinus.

**The Foetal Circulation.**—Blood is carried from the foetus to the placenta by the hypogastric or umbilical arteries, branches of the iliac arteries. It returns from the placenta by the omphalo-mesenteric or vitelline veins. These unite to form the umbilical vein which enters the abdomen and communicates by one branch with the portal vein and liver and by another, the “ductus venosus Arantii,” is continued into the hepatic vein, vena cava inferior and right auricle. The inferior vena cava conveys blood from the umbilical vein, portal vein and lower half of the body. Most of this blood is directed by the Eustachian valve through the foramen ovale to the left auricle. It then passes through the left ventricle and aorta, and part of it is distributed by the common carotid and subclavian arteries to the head and upper limbs. The remainder passes onward and, mixed with venous blood which reaches the aorta through the ductus arteriosus, is distributed to the rest of the body. Thus the head and upper limbs receive a purer supply of blood than the trunk and lower limbs. The blood from the head and upper limbs is more venous than that in the inferior vena cava. It is conveyed by the superior vena cava to the right auricle and ventricle and thence through the ductus arteriosus into the descending aorta. Some of it passes through the pulmonary artery to the lungs and left auricle. From the aorta it passes to the placenta by the umbilical arteries and is there oxygenated.

At birth the placental circulation is abolished, the lungs are inflated with air, the diaphragm descends and the pulmonary artery rapidly dilates. The blood passes from the right auricle through the pulmonary artery instead of the ductus arteriosus. The umbilical arteries from the hypogastric trunk to the umbilicus are obliterated, usually in 2-4 days. The umbilical vein is obliterated from the umbilicus to the portal vein, as well as the branch forming the ductus venosus. The foramen ovale is closed.

**Pathogenesis of Congenital Heart Disease.**—Many of the deformities are due to developmental error in the first two months of foetal life. The defect is generally an arrest of the formative processes by which the various septa are formed, or possibly in the development of the valves and branchial arches. All the great ante-natal developmental processes are completed before the end of the second month and the heart is then almost as well formed as it is a few days before birth. In the intervening period it grows



in size and weight. Hence is it improbable that endocarditis can produce any great structural defect. If it is responsible, it must occur very early in foetal life and during a very limited period of time. Other malformations are often found in the body and mental defects such as mongolism, idiocy and deaf-mutism. More than one child in a family may be affected, e.g., 6 cases of patent ductus arteriosus in one family (de la Camp). The second great cause is endocarditis, due to rheumatism, sepsis or specific fevers affecting the mother during pregnancy. The endocarditis is usually an inflammation of the sclerotic variety but vegetations may occur. It is rare before the fourth month. Both endocarditis and valvular developmental errors are more common on the right than on the left side because of the higher pressure there during foetal life.

If endocarditis causes pulmonary stenosis, it would have to occur before the end of the second month to produce any effect on the ventricular septum. At this early age an inflammatory affection of the pulmonary artery would almost certainly involve the aorta. But the combination of aortic and pulmonary stenosis is very rare. Stenosis is always a process of slow and gradual development and unlikely to have any effect on the development of the septum. Furthermore there is no microscopical evidence of inflammatory action in these stenosed orifices. Yet the association of pulmonary stenosis and patent ventricular septum is extremely common.

Maldeveloped hearts are more susceptible than normal ones to foetal endocarditis. Endocarditis affects the tricuspid valves most often, less frequently the mitral, and rarely the pulmonary or aortic. It may produce changes in the circulation which affect the closure of the foramen ovale or ductus arteriosus.

Errors of development have been ascribed to syphilis, tuberculosis, alcoholism, mental disturbance and injury during pregnancy (p. 21). Moncorvo (1900) found more or less distinct signs of congenital syphilis in 2 out of every 3 cases. Hochsinger only found 7 cases among 500 congenitally syphilitic children. In my experience syphilis is an occasional but by no means constant factor.

**Malposition of the Heart.**—*Ectopia cordis* may occur alone or as part of eventration. It is incompatible with life. The heart lies outside the body, with or without a pericardial covering. The sternum is more or less absent and the skin normal or lacking. In *mesocardia* the heart is in a mesial position, the apex in the epigastrium, and the cardiac sounds are best heard behind the sternum. In *dextro-* or *dextro-cardia* the heart alone is developed on the opposite side. If the viscera are also transposed, it is spoken of *Situs Inversus*. Of this the first case was recorded in 1643 and about 200 since. Probably it is much more frequent than these figures suggest, for the condition is usually only discovered accidentally or post mortem. Two girls with complete transposition and a boy with dextrocardia have come under my observation during two years. In none of them had the



malformation given rise to any disturbance of function. It may be associated with congenital heart lesion and possibly these children are more liable to endocarditis. As a rule it in no way interferes with prolonged life. It is likely to be mistaken for cardiac displacement due to pleural effusion, contraction of the lung and other causes.

**Developmental Errors.**—The *bilocular heart*, normal in fishes, consists of one auricle and one ventricle, and a main vessel arising from the ventricle. The *trilocular* or *reptilian heart* consists of two auricles and one ventricle. There may be a rudimentary septum in the ventricle. Other abnormalities are often present, e.g., patent foramen ovale, auricular septum almost entirely absent, transposition of pulmonary artery and aorta, both vessels situated to the left of the ventricle. The left auricle may communicate with the ventricle only by the blood passing through a patent foramen ovale. More often the right auricle does not communicate with the ventricle. The pulmonary valve is frequently adherent, sclerosed or even calcified. In a boy, aged 4 months, who died from general bronchitis, there was a loud systolic murmur all over the precordium and no cyanosis, a trilocular heart and patent foramen ovale were the only lesions. It is compatible with several years of life. Thus 10 collected cases lived from 11-39 years.

*Patent Foramen Ovale.*—The valvular orifice through which a probe can be passed after death is very common. It is unimportant unless the pressure in the left auricle exceeds that in the right and causes an overflow of blood into the right auricle and secondary venous stasis. Sometimes the valve is completely absent and an orifice of varying size is left. The orifice may be single or cribriform. There may be patency of the orifice with smaller holes in the septum, due to the persistence of the holes in the primary septum. In other instances the septum is membranous and contains numerous orifices. A still rarer condition is the presence of Chiari's net-work, previously described by Rokitansky. It is a fine network in the right auricle extending from the Eustachian valve to the region between the two caval orifices and is not incompatible with prolonged life. The septum may be completely absent, or its inferior part may be absent, and cause inter-communication between the four cavities. Perforations are occasionally found in the lower part of the septum. A patent foramen ovale may give rise to no symptoms. A murmur, if present, ought to be presystolic or diastolic and heard over the centre of the sternum. Sometimes there is moderate cyanosis and a systolic murmur, as in the case of trilocular heart, and probably dependent on other cardiac defects. It is exceedingly difficult to time these murmurs. The diagnosis is rarely possible, though patency can be suspected, if slight respiratory or cardiac affections cause cyanosis.

*Patent Septum Ventriculorum.*—Of this there are all degrees, from a pin-point orifice to complete absence of the septum. It may give rise to no symptoms or physical signs. The septum is rarely perforated from disease in later life. Perforation is usually in the anterior portion, in front of the



infundibulum and between the arterial orifices, that is, in the part known as the undefended or membranous space. It is uncommon as an isolated lesion, being usually associated with pulmonary stenosis and hypertrophy of the right ventricle, less often with other defects. A small perforation may be closed by contraction of the septum during systole. Seeing that the pressure is greater in the left than the right ventricle, the right ventricle may become overloaded and hypertrophied, and the pulmonary second sound accentuated. A difference in pressure in the two ventricles will give rise to a murmur during systole, though such murmur is often absent. The characteristic murmur is harsh, systolic, with its maximum intensity in the third and fourth left interspaces near the sternum or in the fourth left space  $\frac{1}{2}$  in. above the nipple. Its character and site of maximum intensity are by no means constant. Generally it is heard all over the front of the chest, and over the back when the aorta rises partly from the right ventricle. Sometimes there is a marked systolic thrill. The murmur and accentuated pulmonic second sound are the main diagnostic points. If it is associated with pulmonary stenosis, the pulse is practically normal but the pulmonary second sound is not accentuated. In uncomplicated pulmonary stenosis the pulse is weak. Patency of the septum is compatible with prolonged life, but death may result from valvular insufficiency.

*Defects in the Septum of the Bulb* may cause the pulmonary artery to rise from the left ventricle. Transposition of the two main vessels is due to lack of rotation of the septum between them. The aorta rises anteriorly from the right and the pulmonary artery posteriorly from the left ventricle. It is associated with patency of the foramen ovale and ductus, and enlargement of the right auricle and ventricle. It is usually fatal under 1 year of age (in 20 out of 25 collected cases, Rauchfuss), though patients have lived as long as 40 years. One child, cyanosed from birth, died at the age of 7 weeks. Another, with moderate cyanosis and clubbing and a systolic pulmonary and apical murmur, lived to 11 months of age:—the right ventricle gave off a normal aorta from which the great vessels arose by a common origin; the pulmonary artery was somewhat stenosed because of the fusion of 2 of its valves and arose from the left ventricle; the ductus arteriosus was normal, the foramen ovale patent, and a small orifice was present in the interventricular septum. It is difficult to account for the maintenance of the circulation in this case. In an almost similar one, a boy lived to the age of 11 years (Kauffmann, 1906). Pure transposition gives rise to much cyanosis, hypertrophy of the right ventricle, an abnormally loud second sound but no murmur. In other cases the cyanosis varies in type. Older children may have convulsions and attacks of suffocation.

The septum may be completely absent or there may be a direct communication, aortico-pulmonary, of variable size between the two vessels with no other abnormalities. In one such case was a double murmur; in another, a systolic murmur and accentuated second sound. The right.



ventricle is generally hypertrophied. Cyanosis is sometimes present; occasionally patency of the ductus and foramen ovale, patency of ventricular septum, or partial obliteration of the aortic arch between the origin of the innominate and carotid arteries.

*Patent Ductus Arteriosus.*—Premature closure during foetal life causes death of the foetus. Closure after birth is brought about in the following manner. Before birth the duct enters the aorta obliquely and forms a crescentic projection, in consequence of the higher pressure in the pulmonary artery. At birth the pressure rises in the aorta and falls in the pulmonary artery. The wall of the aorta then projects into and partially blocks the opening of the ductus. The descent of the diaphragm pulls down the heart and consequently the ductus is no longer approximately on the same level as the aorta but ascends obliquely to open into it. The flow of blood is obstructed in much the same way as it would be in a stretched rubber tube. Actual closure is due to contraction and thickening of the coats. The duct is functionally impervious after respiration has continued for a few minutes; no longer patent in 7-10 days; and completely closed in 3 weeks, occasionally not for 6 weeks. In premature infants it is often late in closing. It may be partially patent, though blocked in some part of its course. Occasionally it is absent or irregular in its origin, e.g., arising from the left branch of the pulmonary artery. There may be two ducts.

Persistent patency may, or may not, be associated with other defects. As an isolated lesion it is rare. It is due to failure in the normal process of closure, some defect in the relative blood pressures, atelectasis, pulmonary stenosis, disease of other orifices and transposition of the main vessels. The blood pressure may be abnormally high in the pulmonary artery in some of these conditions. In aortic stenosis and atresia the pressure in the aorta is unduly low. A patent duct varies greatly in size and is very susceptible to atheroma. Aneurysm and rupture have occurred.

There may be no *symptoms*, no indication of circulatory disturbance, and no murmur. Cyanosis is almost always absent, for even more blood goes to the lungs. Weak and premature infants have attacks of apnoea and cyanosis, which are often fatal. Symptoms are apt not to develop until the end of the first year or later. The chief ones are palpitations, dyspnoea, inability for severe exertion, and occasionally anginal attacks. Cyanosis, cardiac incompetence and oedema develop in some cases and towards the end.

The physical signs include cardiac hypertrophy, a murmur and a thrill. The right ventricle is enlarged slightly unless the septum is incomplete; for the obstruction to complete systole by an inflow of blood from the aorta into the pulmonary artery, which becomes dilated, gives it more work to do. Dulness in the second right interspace is not uncommon. Gerhardt has described a narrow band of dulness for  $\frac{1}{2}$ -1 in. to the left of the sternum, from the first or second space to the third space. It is due



to dilatation of the ductus. Gibson describes the murmur as accompanying the latter part of the first sound, filling the short pause, accompanying the second sound which may be accentuated in the pulmonary area and is often doubled, and dying away during the long pause. In a child, aged 15 months, who died from ileo-colitis there was no cyanosis or clubbing. A systolic murmur, loudest in the pulmonary area, was heard all over the precordium and behind. The patent ductus was the only cardiac defect. Much importance is attached by some writers to the conduction of the murmur into the upper part of the left interscapular space. As a rule the murmur is loudest over the second, or second and third, left intercostal spaces near the sternum and is transmitted into the vessels of the neck, being louder on the left side. It is prolonged into the beginning of diastole. The murmur in children is rarely of the type described by Gibson. It may not develop for a few months, not until the duct dilates. The thrill is most palpable, when present, in the same situation as that of maximum intensity of the murmur. The pulmonary second sound is accentuated unless the pressure is so great as to limit the opening of the valves. The aortic second sound may be much exaggerated and the closure of the valves cause a palpable diastolic shock in the second left space. Combined systolic and diastolic murmurs, and a single diastolic one have also been described.

The *prognosis* depends on the degree of patency and the presence of other abnormalities. Of 41 uncomplicated cases (Wells, 1908) 12 died under 1 year, 5 more under 10, 4 under 20, and 3 lived over 50 years. Death is due to intercurrent disease or cardiac failure. No doubt the defect is more common than these figures suggest. Cases of recovery have been reported, the duct closing and the murmur disappearing.

*Congenital Closure of Orifices.*—If the foramen ovale is so constricted as to affect the heart, the right auricle and ventricle, pulmonary artery and ductus are abnormally large, while the left side of the heart and the aorta are unduly small. The ventricular septum may be patent and the aorta arise from both ventricles. If the ductus closes before the septum, the pulmonary artery will be atrophied. All the orifices of the heart and main vessels may be stenosed or obliterated.

**Valvular Lesions.**—The auriculo-ventricular valves are rarely abnormal in number, but there may be redundant segments. At times the valves are adherent and the orifice stenosed. A double mitral valve has been recorded. Mitral stenosis is uncommon before 6 years of age. A few cases, probably congenital, have been reported in younger children. In two of them the ductus was also patent. It may be associated with tricuspid stenosis, a defective septum or other defects. Possibly some late cases of mitral stenosis take their origin in a mild congenital affection. Mitral incompetence is still more rare. In one case I regarded the disease as of foetal origin. This was a girl, aged 22 months at death, who had presented all the ordinary signs of advanced mitral regurgitation during life.



The mitral orifice was very large and the cusps were represented by a thick, shrunken, fibrous mass of horse-shoe shape. Some of the chordæ tendineæ were attached to the wall of the ventricle, not to the valves. There was partial adhesion of the aortic valves and one of the tricuspid was a little thickened at the edge. The foramen ovale was closed. Apparently the child suffered from endocarditis during the later months of foetal life. The mother had mitral regurgitation and had rheumatic pains during pregnancy.

Tricuspid stenosis is more frequent than mitral stenosis. The foramen ovale is patent. Regurgitation may be due to faulty development of the valves, which is associated with an unduly large pars membranacea of the septum.

The semilunar valves may be absent or represented by a mere fleshy ring. There may be only 2, equal or unequal in size, and occasionally a third rudimentary one. Sometimes they are redundant, three or four of equal size and one or two smaller ones, or show other variations. The pulmonary are very much more frequently affected than the aortic valves, in the proportion of 109-29 collected cases. No matter the number, they may be adherent, united into a cone and narrowing the orifice to a pin-point, an elongated slit or a small triangle. Sometimes they present cribriform perforations; often they are opaque, thickened, puckered, even calcareous. Subsequent rheumatic and calcareous changes affect the valves more often when they are deficient in number than when they are in excess.

*The Pulmonary Artery and Orifice* are affected in about half the cases of congenital heart disease. Out of 181 cases Peacock found the pulmonary orifice more or less contracted in 90, and the orifice or trunk obliterated in 29. Slight stenosis may be unassociated with other defects. Usually there are found patency of the septum of the auricle and ventricle, one or both. There are three varieties of pulmonary stenosis. The trunk of the vessel may be constricted or show complete atresia, but the latter is very rare. More frequently there is constriction of the infundibular part of the right ventricle, known as intra-cardiac conus stenosis or atresia of the conus pulmonalis. Still more often there is adhesion or malformation of the valves or stenosis of the ostium. Stenosis of the orifice or conus, of varying degree up to complete atresia, is due to contraction of the zone at the outlet of the ventricle, the infundibulum, from thickening of the endocardium or localised muscular hypertrophy. It is more probably developmental than inflammatory in origin. Stenosis, due to adhesion of the valves, is associated with patency of the foramen ovale and ductus, and either enlargement of the infundibulum and right ventricle or narrowing of the infundibulum and patency of the ventricular septum. In stenosis the ductus arteriosus is generally closed, while in atresia it is almost always patent.

The effects of the above conditions on the heart vary with their degree and period of development. In slight stenosis, with perfect septum, the only result is moderate hypertrophy of the right ventricle. This is more



marked if the foramen ovale is patent, and still more if both septa are patent. If the ventricular septum is patent, hypertrophy is greater, the more the aorta arises from the right ventricle. Sometimes the septum is pushed so much to the left that the aorta rises entirely from the right ventricle. In atresia, developing while the ventricular septum is being formed, the right side of the heart is enlarged and the left is diminished. If it develops subsequent to the formation of the septum, the right ventricle, tricuspid valve and orifice are atrophied, the foramen ovale remains patent and the left side of the heart is hypertrophied. The blood is carried to the lungs by a backward stream through the patent ductus, possibly by the bronchial arteries, or by special branches from the large vessels of the aortic arch. There may be a direct aortico-pulmonary communication.

The physical signs are those of hypertrophy of the right ventricle, with a systolic pulmonary murmur and a weak pulmonary second sound. The murmur may be absent in high-grade stenosis, because of the slight blood current and overflow through the septum into the left ventricle. Or it may be transmitted, via a defective septum, into the aorta and carotids. The characteristic murmur is localised over the second left interspace close to the sternum, and is not conducted into the vessels in the neck. A louder one is most intense between the second left cartilage and the xiphoid, and an inch or so on either side. Sometimes its point of maximum intensity is over the pulmonary orifice, occasionally midway between the nipple and sternum at the level of the nipple. It is not conducted much beyond the cardiac area, possibly rather more towards the left clavicle than the right, and sometimes all over the back, being louder on the left side and heard in the left axilla. Carpenter has pointed out that if there is a patent ventricular septum, the murmur will probably be heard in the carotids. In a girl who was born blue, presented moderate cyanosis and clubbing and died at 5 months of age, there was no murmur during life. The pulmonary valves were adherent and left only a pinpoint orifice. No other cardiac defect was present. In a boy of 12 months with extreme stenosis of the conus pulmonalis, the orifice admitting only a No. 1 catheter, a soft systolic murmur was heard in the pulmonary region and to the left of the sternum. It was not conducted into the neck nor heard behind. The ventricular septum was patent and the aorta arose from both ventricles. The right ventricle was much thicker than the left.

A systolic thrill may be also present and most marked in the region of maximum intensity of the murmur. It is by no means constant and is likely to be wanting if the stenosis is extreme. In pure pulmonary stenosis, and possibly if the foramen ovale is also patent, the pulmonary second sound is weak. It is clear and distinct if there is associated patency of the ventricular septum, and accentuated if the ductus is patent. The character of the second sound is only of diagnostic value in early childhood. Later,



hypertrophy of the right ventricle may cause so much blood to flow through the narrowed orifice that the pulmonary tension is increased and the second sound is accentuated. The sound depends greatly on the size of the orifice and the mobility of the valves. Hypertrophy of the right ventricle displaces the cardiac apex to the left. The pulse is larger in volume if the septum is patent than in pure pulmonary stenosis. If the tricuspid orifice becomes incompetent, a regurgitant pulse is visible in the jugulars and the liver is enlarged and pulsates.

The symptoms vary with the degree of obstruction. In the mildest form they may be absent. In others there are cyanosis, clubbing and other evidence of venous stasis. Cyanosis may be present at birth. It is often of high grade and increased by exertion and crying. Paroxysmal cough, dyspnœa, suffocative attacks, fainting, vertigo and fits may be present. These children are easily chilled, stand mental and physical exertion badly, and are very susceptible to infection. Many die from tuberculosis or exanthemata; some from cardiac failure during an attack of dyspnœa or the result of asthenia, a few from cardiac failure and dropsy; and others from infective endocarditis. About one-fifth of the cases of stenosis die in the first year of life and only about one-fourth live over 10 years. Two-thirds of the cases of atresia die in the first year and about 15 per cent. live over 5 years. It is surprising how minute a pulmonary orifice is compatible with life, even though the ductus is closed. The patient may live 50 years or more. A child with complete absence of the pulmonary artery lived for 6 years (Gutkind).

Pulmonary stenosis may be followed by infective endocarditis, destruction of the valves and *regurgitation*; or the valves may contract sufficiently to permit regurgitation. Newton Pitt in Allbutt's "System of Medicine" refers to 99 cases of regurgitation, verified post mortem, of which more than half were due to infective endocarditis. Only 2 were under 10 years of age. A girl, aged 15 years, died after having been under my care for 3 years. Heart disease had been diagnosed in the second year of life. When first seen she presented a thrill and murmur and other signs of pulmonary stenosis. Several months before death the systolic murmur and thrill were replaced by diastolic ones. The diastolic murmur was loudest in the pulmonary region on expiration and in the upright posture, and conducted down the left edge of the sternum. She died after a period of irregular fever and several attacks of hæmoptysis, the last one proving fatal, and infective endocarditis was found post mortem and infarcts in the lungs. The pulmonary valves were alone involved, thickened, puckered and showing warty vegetations.

In another girl, 10 weeks old, blue at birth and always a bad colour, death resulted from bronchitis. A loud systolic murmur was heard all over the precordium and back. There was no thrill. The pulmonary artery arose from the aorta, the ventricular septum was patent, the foramen ovale



fenestrated, and the inferior vena cava opened into the left ventricle. Such a case is impossible of diagnosis.

*The Aorta and Aortic Orifice.*—The aorta may show stenosis or atresia of the conus arteriosus, ostium or valves, complete obliteration of the initial part of the artery or hypoplasia. The signs of *Stenosis* of the Ostium are the same as in the acquired disease. It is rarely present. T. Fisher reported a case in a girl, aged 4 months, the valves being thickened and adherent. A 7-year old boy under my care presented all the signs of aortic stenosis, and heart disease had been diagnosed at 2 years of age, so it was probably congenital. In another boy, aged 14 years, undergrown and free from cardiac symptoms, the physical signs were those of pure aortic stenosis. He died suddenly a week later and the valves were found puckered and adherent, the orifice very small. There was nothing in the previous history to suggest an attack of endocarditis. Both boys had a loud systolic murmur and marked thrill over the aortic area, and a small slow pulse. Friedmann has recorded a similar case in a boy, aged 21 months, the murmur being recognised at 4 months of age.

*Hypoplasia*, sometimes called “congenital stenosis of the arterial system,” is rarely limited to the aorta. About 20 cases are on record, 15 verified after death. It is a uniform and general narrowing of all the vessels usually associated with infantilism, sexual abnormalities and rickets. Its etiology is doubtful and symptoms may be latent until puberty, when compensation begins to fail. The patients are small, weakly and anæmic, easily fatigued mentally and physically, and suffer with palpitations and dyspnœa, sometimes subnormal temperature and œdema. The vessel walls are thin, the pulse weak or absent, and the left ventricle small at first. Later on it hypertrophies and generally ends in dilatation before the age of 30 years. Tuberculosis is common.

*Isthmus Stenosis* or *Co-arctation of the Aorta*, complete and incomplete, is also rare. About 100 cases are on record, only 15 per cent. complete. The sex-incidence is 7 males to 2 females. The isthmus of the aorta is that portion which corresponds with the left posterior aortic root between the fourth and fifth branchial arches, that is, between the origin of the left subclavian artery and the entrance of the ductus. It is thin and narrow during foetal life and develops rapidly after birth. This portion of the aorta may be absent, and the separation between the transverse and descending aorta complete. It may be reduced to a fibrous cord for about a centimetre in length; present a double constriction with a dilated intervening portion; or have an unduly narrow lumen (incomplete co-arctation). The narrowing may take place at the point of entry of the ductus. The constriction is sudden or gradual. Usually the vessel is dilated or funnel-shaped on the proximal side, and thin and small on the distal side.

The ductus may supply or give rise to the descending aorta, and in



still rarer cases is entirely absent. Isthmus stenosis is associated in one-third of the cases with valvular and arterial anomalies and other congenital malformations. The supply of blood to head and upper limbs is quite normal. If the ductus is not patent, the collateral circulation is carried on by anastomoses between the upper intercostal arteries from the second part of the subclavian with the first aortic intercostals; and by the posterior scapular arteries from the second part of the subclavian, the subscapular branches and the internal mammary arteries, anastomosing with the superficial epigastric and posterior intercostals. Cases of mild grade develop no collateral circulation, but the left ventricle is hypertrophied and the aortic second sound accentuated. In the more serious cases the ventricle fails to stand the strain of the anastomotic circulation and death results from cardiac failure. If slight, the condition cannot be diagnosed in early life for the signs develop gradually. Eventually there are present many thick, tortuous arteries under the skin; excessive pulsation in the arteries of the head, neck and upper extremities; hypertrophy and dilatation of the heart, bulging forward of the aorta in the neck; a loud murmur over the precordium, especially over the manubrium, and murmurs in the arteries of the head and upper extremities. If incomplete, a systolic murmur may be heard over the descending aorta between the scapula and the spine. The pulse is exaggerated in the radial artery, and feeble or absent in the abdominal aorta and the femorals, etc.

Clinical symptoms may be absent or limited to dyspnœa, and later on those of cardiac failure. In a case of incomplete isthmus stenosis in a girl, aged 5 weeks, there were no symptoms except slight lividity on crying, and no murmur. Death was due to bronchitis. The ductus was widely patent. The aorta gave off normal vessels and then suddenly narrowed at the isthmus, and the ventricular septum was patent. This condition was quite compatible with life had it not been for the bronchitis. In an adult navvy, seen many years ago, the arterial dilatation and tortuosity of the vessels were very marked, but there were no symptoms whatever. The prognosis varies inversely as the degree of stenosis and directly as the completeness of the collateral circulation. If there is incomplete stenosis of moderate degree, life may be prolonged. The average duration of life is about 30 years. A case of Reynaud's lived 92 years. Rupture of the auricle, ventricle or aorta has occurred, and secondary saccular or dissecting aneurism. The aortic valves are often defective or diseased.

**General Symptoms.**—Cyanosis and clubbing of the extremities are the two symptoms which require most consideration. Less frequently there are noted one or more of the following:—dyspnœa, constant, on exertion only, or in paroxysmal attacks during which cyanosis becomes so intense that the extremities may turn black; attacks of suffocation with loss of consciousness, thready pulse and lividity, lasting up to 15 minutes and perhaps fatal; syncopal attacks; paroxysmal or constant cardiac pain;



a variable, intermittent or irregular pulse; tachycardia; headaches, somnolence, extreme irritability, convulsions and coma; subnormal temperature, cold extremities; delayed physical and mental development, delicate bones, tendency to kyphosis, late sexual development (infantilism). These children feel the cold much, and are liable to "catch cold" and develop tuberculosis, especially in pulmonary stenosis. Symptoms as a whole are most frequent in pulmonary stenosis for it is more compatible with prolonged life. Hæmorrhages may occur. True angina is extremely rare; two cases have been recorded in infants under 6 months of age.

*Cyanosis* or *Morbus Cæruleus* is by no means always present and may be entirely absent, the face being normal in colour, unduly bright-complexioned or presenting an unusual pallor. Sometimes capillary stigmata are seen on the cheeks. If cyanosis is present, it is not necessarily of cardiac origin. When it is, it varies greatly in severity and distribution, may be limited to the extremities or involve the whole body and the mucous membranes. It is always increased by effort, crying and emotional disturbance, and may only be present under these conditions. Paroxysmal attacks of greatly increased severity are not uncommon. The blueness may not develop for some months or years after birth, and is then the result of some illness, such as bronchitis or a specific fever, or because the heart does not grow sufficiently for the growth of the child. When it is present it is almost invariably persistent, though variable in intensity. It is most noticeable in the cheeks, lips, ears and extremities. Some of these infants are plum coloured at first, and the colour may fade or disappear as the child grows. The greater the cyanosis at birth, the worse is the prognosis. Other causes of congenital cyanosis are atelectasis, pulmonary apoplexy, diaphragmatic hernia, cerebral hæmorrhage and sepsis. Two cases in which there was hyperglobulia without heart lesion have been reported by Weil. One of them showed changes in the bone marrow and thymus, and an enlarged spleen. Cyanosis is always due to deficient aëration of the blood and is perhaps aided by the imperfect development of the special affinity of hæmoglobin for oxygen. The blood contains a large increase in the number of red cells and percentage of hæmoglobin; up to red cells 10 million, hæmoglobin percentage 160, specific gravity 1070. The increased number of red cells is compensatory to the increased percentage of  $\text{CO}_2$  in the blood. Other factors sometimes present are venous congestion, and the mixing of venous and arterial blood in consequence of the cardiac defects.

Not much importance can be attached to the mixing of blood, for cyanosis can exist without admixture and complete mixing may occur without cyanosis.

Pulmonary stenosis causes deficient aëration when the stenosis is sufficient to prevent the proper amount of blood being driven through the lungs. Otherwise there is no lividity until compensation fails. General



cyanosis is most marked when the aorta arises from the right ventricle. It is increased by dilatation of the right ventricle and venous stasis, for the blood in the systemic veins becomes still more de-oxygenated. Venous stasis is followed by venous dilatation. There is much liability to hæmorrhage. The retinal vessels are tortuous and distended, causing *cyanosis retinæ* and perhaps neuro-retinitis. A patent foramen ovale will only give rise to cyanosis when, for any reason, such as pulmonary affections or cardiac defects, there is a rise of pressure in the right auricle sufficiently great to drive the blood into the left auricle. Under such conditions the cyanosis develops quite unexpectedly. Out of 77 cases of cyanosis in congenital heart disease the pulmonary artery or orifice was obstructed in 53 (Stille). In a series of 50 cases, cyanosis was present in 23. Of these, 7 out of 13 fatal ones were cyanotic, 2 only on crying, and the defects were pulmonary in 6, viz., transposition of the aorta and pulmonary artery in 2, pulmonary stenosis and regurgitation, atresia of the conus pulmonalis, the pulmonary artery rising from the aorta and the inferior vena cava opening into the left ventricle, pure pulmonary stenosis, and in the seventh isthmus stenosis with patent ductus and septum. The last case only became blue on crying. In 18 out of 37 cases, not verified post mortem, a pulmonary lesion was diagnosed or suspected and 16 showed more or less cyanosis. Cretins are sometimes very blue.

*Clubbing* is most obvious in the terminal phalanges of the fingers and toes, but it is not peculiar to congenital heart disease. It is seen in chronic phthisis, bronchiectasis, prolonged empyema and pressure on the axillary vein. In morbus cordis it is due to the same causes as the cyanosis, does not occur independently thereof, and varies in degree with the severity and duration of the lividity. It is due to congestion, engorgement of the veins and thickening of the vessel walls. There is no increase in the connective tissue. After many years in severe cases a hypertrophic osteoarthropathy, an ossifying periostitis, of the knees and elbows may develop.

*Complications* and *sequels* are numerous. In order of frequency may be mentioned bronchitis and broncho-pneumonia, gastro-intestinal troubles, epistaxis, hæmoptysis independently of phthisis, tuberculosis, albuminuria, jaundice and rarely dropsy; sometimes endocarditis, simple or malignant, occasionally sores about the genitals, anus and extremities, gangrene, rheumatism, chorea, amenorrhœa and metrorrhagia. Sudden death, secondary cardiac failure and cerebral thrombosis may ensue. Out of 449 collected cases of pulmonary stenosis 160 developed tuberculosis of the lungs (Norris, 1904). It may also occur in other forms of congenital morbus cordis.

**Diagnosis.**—Cyanosis without murmur may be due to a mono-ventricular heart, stenosis of the isthmus aortæ and patent ductus, atresia of the conus pulmonalis or irregular origin of vessels. If it is associated with a basic murmur it is due to pulmonary stenosis in 75 per cent., with



or without other lesions, or to multiple abnormalities. In conjunction with an apical murmur I have found it due to transposition of the main vessels or multiple defects. A murmur without cyanosis may be due to a patent interventricular septum and lesions such as are found in acquired heart disease. A diastolic murmur is generally due to pulmonary regurgitation. Considerable displacement of the apex to the left is a sign of grave defects.

Abnormally loud murmurs are almost pathognomonic but congenital *morbus cordis* can exist without murmurs. In comparison with the acquired forms stress must be laid on a loud, harsh or musical murmur, with little or no increase in dulness, and a weak apex beat; increased dulness to the right; no apical murmur; a weak pulmonary second sound in pulmonary stenosis; and the evidence afforded by X-ray examination. Functional pulmonary murmurs are common during life. A diagnosis of pulmonary stenosis and phthisis must be accepted with caution unless verified after death.

**Prognosis** depends on the symptoms rather than the physical signs; on the nature of the defect, whether developmental or inflammatory in origin; upon its effects on the heart and circulation; the maintenance and duration of compensation; and the effect on the growth of the child. A loud murmur is quite compatible with normal life, while a fatal issue may ensue very early, although there may be no murmur and indeed no evidence of cardiac defect. Patients become more or less used to cyanosis, but as a general rule the prognosis is worse in proportion to the degree of lividity. Cyanosis may prove fatal from syncope, fits, coma or secondary lung trouble. Indications of a guarded prognosis as regards duration of life are the evidence of cardiac dilatation and hypertrophy, especially of the left ventricle, unduly frequent pulse, shortness of breath or attacks of dyspnoea, with or without exertion, and delayed physical and mental development. Convulsions, paroxysmal dyspnoea, persistent low temperature and hæmorrhages are bad signs.

Periods of stress are likely to arise from pulmonary affections, whooping cough, gastro-intestinal troubles and other ailments. Murmurs may disappear entirely, or disappear and return during respiratory distress. At puberty undue stress is thrown upon the heart by reason of the cardiac and vascular changes. Nevertheless I have seen no special ill-effect in these cases at this time of life.

Peacock's statistics show that out of 181 cases 119 presented defects of the pulmonary artery, orifice or conus; and that 86 per cent. of the cases of cyanosis which survived the twelfth year had pulmonary stenosis. Out of 64 with pulmonary stenosis and patent septum 14 lived over 15 years. Of 20 in which the stenosis was associated with a patent foramen ovale, 11 lived over 15 years, 1 to 57 years. The average duration of life of 28 with an impervious pulmonary artery was 3 months; 3 lived to 9 or 10, and 1 to 12 years.



In bilocular heart death ensues in a few days. A trilocular one is compatible with adult life. Simple patency of the ventricular septum may be quite unimportant for the orifice may be closed during systole. Some patients live a remarkably long time though the defect is serious. As a whole it is rare for the subjects of congenital heart disease to live long. About one-quarter die in the first year, half in the first 5 years, three-quarters in the first 15 years, and only 10 per cent. live over 30 years.

The social and hygienic surroundings of the child must be taken into consideration because of the liability to rheumatism and phthisis. Muscular exertion and privation are often injurious and lead to failure of compensation. The susceptibility to secondary infective endocarditis must always be remembered.

**Treatment.**—Little can be done beyond bringing the child up under the best conditions of life available, and treating cardiac failure and complications as they arise. The child must be warmly clad and well cared for generally. It is a mistake to bring these children up as chronic invalids. Let them live the ordinary life of childhood, as far as is compatible with the nature of the mischief and the effect on the general health. Provided the child grows at the normal rate and there is no circulatory disturbance, no evidence of mischief beyond the murmur, little attention need be paid to the local trouble. If there is much hypertrophy, over-exertion must be guarded against. Protect them from chills and exposure to infection, from excitement and outbursts of passion, and from errors of diet.



## CHAPTER XLII.

### ACQUIRED HEART DISEASE.

*The Myocardium—Cardiac dilatation—Endocarditis—Valvular defects—Pericarditis and effusion—Mediastino-pericarditis—Adherent Pericardium.*

The muscular walls of the heart are affected by hypertrophy, dilatation, and various forms of inflammation and degeneration. The common causes of hypertrophy of the left ventricle, in order of frequency, are mitral regurgitation, adherent pericardium, aortic stenosis and regurgitation, and chronic interstitial nephritis. Hypertrophy of the right ventricle is present in many forms of congenital heart disease and various causes of increased pulmonary tension, e.g., pertussis, chronic bronchitis and other lung affections, mitral stenosis and mitral regurgitation. Undue exposure of the surface of the heart from retraction of the lungs, rickets or other causes must not be mistaken for hypertrophy.

*Overstrain due to Exercise.*—The ceaseless activity of children, notably boys, causes moderate hypertrophy. Both sexes have a special aptitude for short spells of active exercise and are unfitted for prolonged fatigue. Long distance bicycling, walking, paper-chases and similar exercises may produce serious or even fatal illness. The paper-chase must not be entirely condemned, for the boy is not compelled to run the whole time or the whole distance. After such a strain he may be merely tired ; or may faint or vomit by the way and be obliged to lie down and rest ; or may vomit all the evening after ; or become insensible while running and remain so for hours ; or die while running (Dukes). Two factors are in action, viz., fatigue and auto-intoxication by the products of active metabolism. They may produce dilatation of the heart, acute or gradual in onset ; or simple hypertrophy and dilatation. This is most apt to occur when the heart muscle is already weakened by some toxin, such as that of influenza, measles or an infectious cold.

Vomiting is the most important sign. The symptoms of overstrain are anæmia, languor, shortness of breath on exertion and perhaps a sharp pain at the heart. Examination shows dilatation, increased frequency and irregularity of the heart and pulse, and perhaps a murmur. The ordinary signs of over-exertion are inability to eat a proper meal, disturbed sleep, and shortness of breath on exertion.



Fatalities from overstrain are rare. The boy recovers with complete rest for a few weeks and careful supervision for some months after. The younger the child, the better is the prognosis, but there remains a liability to recurrent dilatation. After puberty irritability of the heart, cardiac pain, palpitations and dyspnœa may persist for months or years. Gentle exercise must be resumed after a time. Smoking and alcohol are injurious.

Exercises for children should be interesting rather than formal, bearing in mind that it is the pace that kills. Children should be allowed to walk, not made to walk, and the distance must be limited. Streets are unsuitable. Croquet, bowls, easy tennis, bicycling on level ground, golf and other mild games are the most suitable, if for any reason the amount of exercise has to be limited. The child must rest for half an hour before meals, especially before dinner.

**Dilatation of the Heart** occurs with exceptional ease in children and is most serious when it is on the right side. It is the result of diminution or loss of muscular tone, and work beyond the power of the impaired muscle. The output of blood from the ventricle is remarkably constant but the peripheral resistance varies. The muscle has much power of accommodation. The ventricle does not empty itself completely at each systole and the amount of residual blood varies with the degree of diastolic distension, and this depends on the tonus of the muscle and increases *pari passu* with diminution of tone. Tone is greatly dependent on the nervous system. If it is insufficient to cope with the distensile stress of intra-ventricular pressure, dilatation results. The dilatation often comes on suddenly and subsides almost as suddenly. It is irregular in its development, rather than steady and continuous, showing that it is not due to impaired contractile power. In cloudy swelling and myocarditis both contractile power and tone are defective.

Acute dilatation is apt to occur during influenza and prove rapidly fatal. Within 24 hours, it is due to the action of the toxin on the bulb. The pulse is very frequent and often intermittent; respirations reach 60-70 per minute; and there is an increase in the area of cardiac dulness. Cyanosis and coldness of the extremities may be present. In about 12-72 hours the symptoms subside and the temperature falls to normal. I have known cases in babies in which tachycardia has been the only sign.

A more common and more dangerous type of dilatation, acute or chronic, is seen at a later stage of influenza, diphtheria, rheumatic fever, scarlatina, nephritis, pneumonia and pertussis, or other infective disorders. It is due to the direct action of a toxin on the cardiac muscle, producing cloudy swelling or myocarditis. The pulse is weak, slow, irregular and often intermittent. Precordial or angina-like pains are not uncommon; and there are marked symptoms of cardiac collapse, with attacks of faintness or syncope which may prove fatal. Such a condition may follow the ordinary infectious cold. Dilatation also occurs in malnutrition, local or



general, when an extra strain is thrown on the heart by increased muscular exertion, raised blood pressure, and cough. It is due also to valvular disease and pericardial adhesions. In some of these affections the dilatation affects all the cavities of the heart, while in others it is limited to the right or left ventricle chiefly, if not entirely. More than one of the different factors, viz., malnutrition, toxæmia and mechanical strains, are active in some of the cases. For instance, in pertussis all are involved.

In acute dilatation we find marked increase in cardiac dulness, a feeble and diffused apex beat, faint first sound at the apex, accentuation of the aortic and pulmonary second sounds, murmurs, dyspnœa, weak pulse, precordial pain, and tendency to collapse. Mild cases show merely irregularity of the heart and shortness of breath. In more severe ones there are dyspnœa, distress and sleeplessness. Bronchial breathing over the base of the left lung is not infrequent. Anorexia, gastralgia, nausea and vomiting, and diarrhœa or constipation, are more common than purely cardiac symptoms. In cardiac insufficiency dilatation of the right ventricle, tricuspid regurgitation and anasarca are less common than in adults. The liver is early and much affected, and breathing more than the pulse. Pulmonary catarrh and deficient urine are earlier signs than anasarca.

**Myocarditis** is a common complication, perhaps the only sign, of rheumatic fever and liable to occur in diphtheria, specific fevers and infective disorders. It is present to a slight extent in endocarditis and still more in pericarditis. In the absence of these affections it is often overlooked or mistaken for simple dilatation.

Myocardial disease is primary, due to the action of toxins, or secondary to cloudy swelling or inflammation. The physical signs, symptoms and prognosis are much the same in each case, and it is not always possible to make an exact clinical diagnosis of the state of the cardiac muscle. *Cloudy swelling* is not strictly inflammatory. It occurs in infective disease with high fever, notably acute rheumatism, typhoid fever and diphtheria, and is due to the action of toxins. The muscle may be soft and friable as early as the fourth day. The heart, especially the left ventricle, is dilated. The granules in the muscle fibres are soluble in acetic acid and in caustic potash. They are not fatty, but on their way to become fat. *Acute segmentary degeneration* is sometimes a further stage. *Myomalacia cordis* is rare in childhood and depends on coronary obstruction and disease. *Fatty degeneration* is very apt to occur in diphtheria, scarlet fever, typhoid and pertussis. It has been produced in animals by the injection of diphtheritic albumoses (Sidney Martin). Waxy degeneration and Zencker's type are very rare.

Myocarditis is acute or chronic, interstitial or parenchymatous, diffuse or circumscribed. The acute interstitial type is due to osteomyelitis, septic scarlatina and other infections. It is almost invariably focal, when due to pyæmia, and may terminate in an abscess which bursts into the pericardial



cavity or through the endocardium. The focal lesions may consist of organisms and round cells, and are not necessarily abscesses. The chronic form is of syphilitic origin, or secondary to valvular disease or adherent pericardium. It produces fibroid or sclerotic patches, "scar of the heart," in the myocardium. The diffuse form is generally syphilitic. The term, Chronic Myocarditis, is sometimes used to include all chronic myocardial degenerative affections, whether fatty, fibroid or due to coronary obstruction. These are more strictly results. Pericarditis does not extend to the myocardium as an acute interstitial inflammation, but causes some cloudy swelling and segmentary degeneration of the underlying muscle. A true interstitial myocarditis, with or without pericarditis, is rare in rheumatism.

The parenchymatous type is almost always a cloudy swelling, with little or no interstitial inflammation, and secondary fatty degeneration. It is the most important form of cardiac inflammation in the young, chiefly seen in acute rheumatism (q.v.). Irregularity of the pulse is the first sign. It is extraordinary how slight and indefinite the symptoms may be. Perhaps there is only a little restlessness at night, impaired appetite, disinclination for exertion and an evening rise of temperature. The child may look quite well, a little pallid or slightly livid. Occasionally there is cough and definite shortness of breath, increased by exertion. Dyspnœa may be severe, and associated with coldness and lividity of the extremities. Rapid emaciation may be the chief symptom.

On examination the heart is found dilated, the degree of dilatation varying with the severity of the case. The apex beat is displaced outward, perhaps not palpable; the impulse diffused and weak; dulness extends one or more finger-breadths beyond the apex and to the right of the sternum, and even up to the third rib; the first sound at the apex is short and weak and may be replaced by a murmur, and the second sound is sometimes inaudible, or the sounds are foetal in character. A murmur is by no means constant, even in great dilatation. It is usually systolic, rarely presystolic. The absence of a murmur may lead to neglect of complete examination of the heart, the non-recognition of the dilatation, and a failure in diagnosis. The dilatation affects all the cavities or may be limited to the left ventricle. The pulse is increased in frequency, soft and irregular, sometimes very feeble and very frequent.

Post mortem the cavities and orifices of the heart are found dilated, the lung displaced and the left lower lobe compressed. The amount of fatty change is variable. Naked eye appearances show little or no change. In the rheumatic type Aschoff has found submiliary nodules in the sub-endothelial and sub-pericardial tissues in the neighbourhood of the arterioles. *Chronic myocarditis* is indicated by arrhythmia, bradycardia, irregular breathing, and greatly increased frequency of pulse and respirations on slight bodily or mental excitement.



*Diagnosis* is difficult for it may be impossible to differentiate the inflammatory affection and its resulting dilatation from the dilatation due to loss of muscular tone and degenerative changes. If, however, dilatation is found in the course of rheumatic fever, or in the absence of any exciting cause in a child, it must be regarded as probably due to rheumatic myocarditis and treated accordingly. Much dilatation can occur in mild diphtheria and occasionally in septic throat affections. Limitation of the mischief to the left ventricle is in favour of diphtheria as the cause.

The *prognosis* depends on the cause, degree of dilatation and the treatment. It is better in rheumatism than in diphtheria or influenza. Provided the affection is rheumatic, that the child is seen early before the dilatation is extreme, and that complete rest in bed for 6 weeks or more is insisted on, the patient will recover. In the degenerative cases, such as diphtheria, the more the dilatation extends beyond the nipple line, the greater is the danger. Sudden death may occur at any time, even in moderate dilatation. It is more common in diphtheria than rheumatism. Sudden death in the course of myocarditis must be distinguished from death due to paralysis of the vasomotor centre in the bulb and profound fall in blood pressure, with extreme dilatation of the vessels in the splanchnic area. This is characterised by rapid fall in blood pressure, weak and empty pulse, pallor and collapse, in the course of an infective disease. It may come on with appalling suddenness, the child sitting up in bed and falling back dead. Sometimes death is preceded by a period of profound collapse, dilated pupils and feeble cardiac action, the respiration being little or not at all affected. No change is found in the cardiac muscle after death. Bulbar palsy, in the course of diphtheria, etc., is closely allied. It causes cardio-pulmonary paralysis, dyspnoea and rapid death. In a milder type it produces dyspnoeic attacks, named by Guthrie "Bulbar Crises." Endocarditis, and pericarditis still more so, increases the gravity of the case. Vomiting, coldness and collapse are bad signs. The pulse rate is a reliable guide.

*Treatment.*—Complete rest is of vital importance, with careful feeding, the avoidance of all foods liable to cause indigestion, and freedom from excitement and worry. Rely on digitalis in all cases of insufficiency of cardiac muscle but remember that it is cumulative and must not be given for long periods at a time. There is no contra-indication in the shape of high blood pressure. Stimulants are necessary, and occasionally oxygen inhalations and possibly venesection. An ice bag over the precordium is sometimes useful. Camphor is given for fainting and collapse; and sparteine sulphate, strychnia and other such drugs.

**Endocarditis** is a more insidious disease in children than in adults and almost the sole cause of valvular mischief after birth. Often it produces no symptoms until well advanced. It may be present in the newborn. Apart from congenital cases it is rare under 2 years of age, and uncommon before



the fifth year. It attacks mainly the valves exposed to the highest tension and therefore liable to injury. Hence it is most frequent on the right side before birth and the left after birth ; and in children most often affects the mitral valves, the aortic being less liable to injury by reason of the low blood pressure in childhood. From 60-80 per cent. of the attacks are due to the poison of acute rheumatism ; and the remainder to chorea, tuberculosis, scarlatina, measles, diphtheria, pneumonia and, rarely, other infections. Not infrequently it is impossible to find a history of any antecedent disease as a probable cause. In such cases a family history of rheumatic fever or morbus cordis is suggestive of a rheumatic infection. In specific fevers the endocarditis may occur with or without articular symptoms. It may follow scarlet fever long after the fever has passed away and without any pyrexia. Post-scarlatinal rheumatism is closely allied to true rheumatic fever.

The valves and tendinous cords are swollen and infiltrated. On the endocardium may be a deposit of lymph which is subsequently organised into fibrous tissue. As it contracts it causes adhesion, stiffness, thickening and puckering of the valves ; thickening, shortening and sometimes rupture of the cords. Three types are found post mortem :—(1) Verrucose, in which there are vegetations of various sizes. The small nodules often found on the valves of the newborn must not be mistaken for vegetations. In chorea, and sometimes in rheumatic fever, the valves are slightly affected ; a row of minute granulations being found on one or more valves. (2) Sclerotic, in which there is contraction of the valves and orifices. (3) Ulcerative, affecting both the valves and walls of the cavities. Endocarditis can be subdivided into two groups. In the first, or rheumatic group, a secondary infection may lead to the development of the second or septic form, liable to become ulcerative. The septic variety may be primary, in that it is not preceded by a rheumatic infection or a damaged valve, but it is secondary to some local source of infection in the throat, lungs, bones, etc. The common organisms are the streptococcus, staphylococcus, pneumococcus and, less often, the gonococcus, influenza bacillus, *B. coli*, etc.

*Infective Endocarditis* is uncommon in the young and runs the same course as in adults. There is no sharp line of distinction from acute endocarditis. The name is generally limited to cases due to pyogenic organisms, and ulceration is not invariable. Nor is it always fatal. In most instances it is secondary to rheumatic endocarditis or congenital heart disease. Cases are septic, typhoidal, cerebral or cardiac in type, according to the predominance of the particular symptoms.

*Symptoms.*—There may be no signs of the endocarditis at the onset ; or perhaps a little irregularity of the pulse and slight rise of temperature, occasionally precordial pain and some breathlessness on excitement or exertion. There is little or no increase in dulness for blood pressure is low and the cardiac muscle resistant. Slight dilatation of the left auricle is difficult to recognise. The cardiac first sound at the apex, in mitral disease,



is weakened or impure. Presently a murmur develops, localised for a time and then extending. It is more readily conducted than in adults and of higher pitch, accentuated by crying, unaffected by breathing, and constant. It may become musical quite early. Rapid breathing and râles interfere with its recognition. The most common site is over the left ventricle, perhaps it is best heard in the third left interspace near the sternum. It must be differentiated from venous bruits, hæmic murmurs and cardio-pulmonary ones.

In the infective type we find anæmia, greater fever, more frequent pulse and respiration rates, irregularity and intermittence of the pulse, sweating, emaciation, vomiting, purpuric eruptions, embolic infarction, diarrhœa, melæna, hæmatemesis, albuminuria and hæmaturia, cardiac pain and syncopal attacks. The fever is septic or typhoidal in type, rarely running the low irregular course often seen in adults. The heart is enlarged; and the murmurs vary from day to day, are more or less constant, or may be absent. Foetal heart sounds and gallop-rhythm may be noted, if there is no murmur. The blood shows leucocytosis. The liver and spleen are enlarged. Pulmonary catarrh, pleurisy, infarction and hæmoptysis may occur; and other signs, varying with the distribution and infectivity of the emboli.

The autopsy may show recent endocarditis, adherent pericardium, endocarditis and ulceration of the valves, exuberant granulations on the valves and endocardium (in one child they were limited to the surface of the auricle) up to the size of a small marble, perisplenitis, enormous spleen (e.g., 15 oz. in a boy, aged 13 years), and infarcts in various organs, some of which may be red, others firm and white or softening and breaking down. In a girl, aged 13, a secondary abscess was found in the right corpus striatum, although infarcts in the spleen showed no sign of necrosis. Another girl, aged 15, died from pulmonary hæmorrhage, due to infarction. The diagnosis of the infective character of endocarditis is based on the course of the case and the presence of some of the above symptoms in combination, especially a pyæmic type of fever associated with morbus cordis and indications of infarction. The prognosis is not absolutely hopeless. Judson (1905) reported a case of recovery from the pneumococcal form, in a boy aged 10. In 1891 I recorded the recovery of a boy, 21 years old, with tricuspid regurgitation, anæmia, large spleen, profuse sweating, and a hectic temperature for 2-3 months. The outlook depends on the virulence of the infective organisms. Treatment is symptomatic. An attempt should be made to isolate and cultivate the organism, and prepare and give a vaccine.

*Pneumococcal Endocarditis.*—The endocarditis present in 1 per cent. of all pneumonias and 4-5 per cent. of fatal cases is not always due to the pneumococcus. This organism is responsible for  $\frac{1}{4}$ - $\frac{1}{5}$  of the cases of infective endocarditis. The disease is twice as common in females as in males; rare under 30 years of age; and generally affects the aortic or mitral valves, or



both, being much more frequent on the left side. The vegetations are usually massive. It may occur before, during or after the lung mischief. Netter found it associated with pneumonia and meningitis in 45 out of 63 cases. The pulse is rapid, leucocytosis often absent, and the organism can be grown from the blood. It lasts from a few days to months, and is generally severe or malignant.

*Tuberculous Endocarditis*.—Minute vegetations are sometimes found on the valves, chiefly the aortic and mitral, of children dying from miliary tuberculosis. A true tuberculous endocarditis is a very rare condition. The bacillus is often present in the blood stream, but the endocardium and the lining of the blood vessels are insusceptible to infection. Tuberculous arteritis starts in the adventitia, and it is probable that the rare tuberculous infection of valves is through their blood or lymph supply. Anatomically it is like other forms of infective endocarditis; and is vegetative, verrucose, fungating and ulcerative. The bacillus is present but no giant cells. The mere recovery of the organism from a vegetation is no certain proof that it is the causative agent. It may be merely superimposed.

*Tuberculosis of the Myocardium* was mentioned by Laennec in 1826 and recorded by Townsend in 1832. It has been described as a primary disease, though its existence in this form is not generally accepted. Nearly half the patients are under 15 years of age. Both sexes are equally liable. Usually the primary source is a tuberculous bronchial or mediastinal gland, and many owe their origin to extension from the pericardium, while the rest are a part of a general tuberculous blood infection. Miliary tubercles are found underlying the endocardium, most frequently in the ventricles. Large tubercles may be formed in the auricle, ventricle or inter-ventricular septum. They vary in size from that of a millet seed to a hen's egg, are yellowish in colour, may have a caseous centre, and closely simulate gummata. Sometimes this variety is associated with interstitial myocarditis. This may occur alone, due to diffuse infiltration with round cells and connective tissue, giant cells, caseous areas, and no tubercle bacilli. Such a case was reported in a boy, aged 3 years (W. M. Smith, 1902). It is comparable with tuberculous cirrhosis of the liver. A diffuse tuberculous infiltration, caseous in character, is a rare sequence of tuberculous pericarditis. The chief symptoms are dyspnoea, gallop-rhythm, tachycardia or bradycardia, arrhythmia, palpitation and cyanosis. They may be entirely lost in those of the general disease. A gumma is more grey in colour and more sharply circumscribed. No inflammatory nodule in the heart should be diagnosed as a gumma, unless there are other signs of syphilis and no evidence of tuberculous disease.

*Tuberculosis of the Pericardium* is due to extension from a caseous gland, especially the pretracheal gland; occasionally from the lungs, pleura, myocardium or bones; rarely part of a general tuberculosis or a tuberculous polyserositis. In general miliary tuberculosis miliary tubercles



are scattered about the surface, chiefly at the base. In the other varieties there is an infiltration and great thickening of the pericardial layers, with obliteration of the sac. The heart may seem to be ensheathed in a sort of cuirass which to the naked eye does not suggest tuberculosis. Sometimes it sets up a serous, sero-fibrinous, purulent or hæmorrhagic pericarditis. In rare instances it is primary and acute in onset. Usually there is an interstitial myocarditis as well. The chief sequels are adherent pericardium, ascites, nutmeg liver, cyanosis, polyserositis, and sometimes mediastinitis. The disease is chronic, slowly progressive, and ends fatally in 4-8 months.

**Valvular Disease** is the sequel of endocarditis, omitting those cases which are of developmental origin. It is impossible to give accurate statistics of the frequency with which the different valves are affected, and of the relative frequency of valvular affections to other cardiac inflammations for constantly two or more varieties are combined. Taking cardiac diseases as a whole, the order of frequency of the different affections is simple dilatation, myocarditis, endocarditis and valvular lesions, pericarditis, pericardial effusion and adherent pericardium. The mitral valve is involved in 4 out of every 5 cases, and in three-quarters of these there is simple regurgitation.

*Mitral regurgitation* produces practically the same signs as in adults, except that there is less tendency to anasarca, œdema of the lungs, nutmeg liver and ascites, though all these develop in late stages. In other words there is greater compensatory power in the heart muscle. The pulse is generally regular, and arrhythmia is rare under 12 years of age. The left ventricle hypertrophies readily and leads to the production of the *cor bovinum*. The area of dulness and pulsation is large, the apex displaced outward and downward, the impulse heaving, and the right ventricle rests on the diaphragm and causes epigastric pulsation. Usually the child is pale and wasted, and the chief symptoms are referable to the digestive organs. The character and mode of conduction of the murmur are valuable aids to diagnosis and prognosis. When the left auricle and ventricle are of normal size, the cardiac sounds are not heard behind, for lung tissue is between them and the posterior thoracic wall. If these cavities are dilated, the lung is partly compressed or pushed aside, and the heart comes more or less into contact with the posterior thoracic wall and the bodies of the vertebræ. Under such conditions the murmur of mitral regurgitation is conducted down the left side of the spine, and the degree of conduction and loudness of the murmur are a fair measure of the dilatation and the amount of regurgitation. Simple loudness of the murmur in the interscapular region, without conduction downward, suggests a dilated and failing left auricle. A loud murmur may reach the head, sacrum and extremities. The character of the pulmonary second sound is of very great assistance. If it is loud and ringing, it indicates high pulmonary tension and considerable regurgitation. And the first sign of cardiac failure is



some weakening in the loudness of this sound. Dilatation of the right ventricle is a sequel of the hypertrophy developed to overcome the increased pulmonary tension.

*Mitral stenosis* is rarely congenital, seldom detected under 6 years of age, and not very uncommon in youth. A marked case in a 3-year-old boy, due to rheumatism, ended fatally from chorea. It is more frequent in girls than boys. The button-hole orifice is rare. Generally it is funnel shaped, from adhesion of the edges of the valves. It is often conjoined with mitral regurgitation, or a sequel of it, and may be present without a murmur, even in the worst cases. The murmur is auricular systolic in time, generally presystolic, sometimes mid-diastolic. An early diastolic murmur is due to the suction action of the diastole of the ventricle. Increased dulness in the third left space shows enlargement of the left auricle. The second sound at the base is often reduplicated and the pulmonary second sound always accentuated. At the apex the first sound is loud, clear, abrupt, banging or thumping, perhaps a regular thud; sometimes reduplicated. The heart's action is cantering. The pulse is very variable. If the right ventricle remains sound and the degree of stenosis is small, the pulse is about normal. If the orifice is very small, it becomes very small, weak and irregular, because the left ventricle receives a scanty supply of blood. The more frequent the pulse rate, the greater is the failure of the right ventricle. The failure of development of the left ventricle produces general wasting and maldevelopment of the body. So much is this the case that delayed growth in morbus cordis is strong evidence of mitral stenosis. The facial aspect is a curious dusky pallor. The backward pressure in the lungs leads to pulmonary catarrh, hæmoptysis, congestion and collapse of the lobules, cell proliferation and diapedesis, and brown induration of the lung. Later on the ordinary signs of backward pressure develop but it is not accompanied by ascites or anasarca, unless there is also mitral regurgitation and tricuspid regurgitation. The chief symptoms are dyspnœa, digestive troubles, sometimes epistaxis or hæmoptysis; and the effects of emboli, carried to the lungs from small fibrinous clots forming in the right auricle or ventricle, or from the left auricle to the brain, spleen, kidneys and, rarely, the liver.

*Tricuspid stenosis* is rarely congenital. It is most common in girls, whereas congenital morbus cordis affects both sexes equally. It is almost always associated with mitral stenosis, and often with aortic stenosis as well. Its etiology is the same as that of endocarditis of the mitral valve. Dyspnœa on exertion is the most constant symptom. Finally dyspnœa occurs when the child is at rest, and orthopnœa, paroxysms of urgent dyspnœa and cyanosis, Cheyne-Stokes breathing and precordial pain. Cyanosis, sometimes clubbing of the fingers, and dropsy may be present. In pure tricuspid stenosis the child is anæmic. If there is mitral stenosis as well, we find catarrh of the lungs, hæmoptysis and the dusky facial aspect.



Subjective symptoms are constantly absent or due to cardiac failure. The veins in the neck are much dilated and show auricular pulsation. In pure stenosis the ventricular pulse in the veins should be absent. The cardiac defects vary in accordance with the degree of stenosis, the purity of the lesion, or the presence of stenosis of other orifices. The right ventricle and auricle are dilated and sometimes hypertrophied, but not so markedly as is the left auricle from mitral stenosis, for there is less resistance to backward pressure in the great veins. The amount of dilatation and hypertrophy of the right ventricle depends on the presence and extent of mitral stenosis. The disease is often overlooked because the signs are masked by those of mitral stenosis. The physical signs are a presystolic thrill and murmur, with evidence of dilatation of the right auricle, pulsation in the jugular veins, absence of ventricular pulse in the jugulars, and pulsation of the liver. Pericarditis, adherent pericardium and infective endocarditis are apt to ensue. The congenital cases live from a few weeks to a few years. The acquired disease is most frequently fatal in the third or fourth decade of life, at an average age of 35 years.

*Tricuspid Regurgitation* is the sequel of failure of the right ventricle from any cause, chiefly mitral and pulmonary valvular affections or lung diseases, including asthma. It gives rise to the usual signs of backward pressure, viz., dilated veins in the neck with auricular and ventricular pulsation therein, nutmeg liver, pulsation of the liver, enlarged spleen, ascites, anasarca and cardiac kidneys. The murmur is systolic, loudest at the junction of the fifth right cartilage with the sternum, conducted upward along the left edge of the sternum, outward to the right and toward the apex, being gradually lost as it nears the apex and accentuated again by the mitral regurgitant murmur, if there is mitral incompetence. In the few cases in which it is heard behind, it is conducted to the right of the spine.

*Aortic Stenosis* is rarely found in a pure form. It is occasionally congenital. In the acquired type it is generally associated with aortic regurgitation. Mitral regurgitation is a common sequel, either due to secondary dilatation of the ventricle or an associated endocarditis of the mitral valve. The murmur is often heard over the back, louder over the upper than the lower dorsal vertebræ, the reverse of what is the case in the murmur of mitral regurgitation. *Aortic regurgitation* is exceptional as an isolated lesion in early life. Seiffert has noted it at the age of 18 months. Two cases have been under my care. The physical signs and course are the same as in later life, except in so far as the lesion is not a progressive degenerative one and good compensation may be established and maintained.

*Prognosis in Heart Disease.*—Much too grave a view has been taken in the past, and indeed is still prevalent, of the serious outlook in all forms of cardiac disease. Both acute endocarditis and pericarditis are more likely to end fatally in children than in adults, because of the myocarditis which is so often present in addition and the consequent tendency to dilatation and



cardiac failure. The cardiac muscle has not the powers of resistance to strain which it attains in later life. Children succumb during an acute attack from rapid dilatation. Or gradual dilatation, with cardiac failure and dropsy, may ensue within a few months. Similarly the prognosis in rheumatic fever is more grave on account of the liability to myocarditis and endocarditis. The possibility of subsequent attacks makes the prospect worse, for each illness is likely to accentuate the cardiac mischief. Endocarditis in chorea is not of serious import. The duration and severity of the chorea do not appear to influence the severity of the endocarditis.

As a general rule the prognosis of valvular disease is less serious than in adults. Before puberty compensation is much more readily established and does not break down. High arterial tension is comparatively very rare in early life. Even children, who have presented serious symptoms and had several valves involved, may recover and lead a long and active life. A boy, aged 14, developed double aortic and mitral regurgitant murmurs during an attack of rheumatic fever. He subsequently lived an active life at the bar, bicycled freely, and in spite of hard drinking bouts, with *mania a potu*, lived to 48 years of age. Compensation in childhood is not likely to break down from severe mental strain or emotional distress. Physical strain is less severe, and the common causes of cardiac and arterial degeneration, syphilis and chronic alcoholism, are wanting.

If hypertrophy cannot take place at a rate equivalent to that of bodily growth, the prognosis is bad. An adherent pericardium is of very grave import and must be regarded as the most serious cardiac affection of early life. The heart cannot hypertrophy efficiently and breaks down at the periods of rapid growth, notably at puberty. This is a critical time of life in most cardiac cases, for compensation constantly remains good until then. All lung affections, by throwing an extra strain on the musculature of the heart, aggravate valvular lesions; so, too, anæmia and any prolonged illness by their effect on the cardiac muscle. A fatal result may ensue from wasting and increasing weakness, with few or no signs of backward pressure. Dropsy, albuminuria and cardiac irregularity may all be absent.

In valvular disease the prognosis depends on the cause, the valves and number of valves affected, the nature of the lesion, the character of the murmur, the effects on the heart and circulation, and the effect on growth. Is the lesion traumatic?—i.e., due to injury, strain or an acute disease which sooner or later comes to an end. If so, the primary mischief ceases as an active process, although the mischief already done remains. This is the usual position in childhood. Rheumatic fever is the common cause and ceases. The valves or orifices are, however, liable to further contraction, or to secondary infection and infective endocarditis. In congenital disease the mischief is not progressive and the whole question of prognosis comes to depend on the other factors mentioned.

The prognosis of stenosis is always more grave than that of



incompetence, especially at the auriculo-ventricular orifices. Mitral stenosis causes less distress than incompetence, but the heart is more liable to break down under extra strain and there is greater liability to embolism. The tendency of stenosis is to gradual increasing obstruction, actual or relative, for the orifice cannot grow with the heart. In dealing with mitral incompetence we have to decide whether it is structural, due to partial destruction or contraction of valves, or dependent upon general dilatation of the cavities and orifices. A structural defect is more serious than relative incompetence. Sometimes the murmur of mitral incompetence is replaced by that of stenosis, by reason of gradual contraction of the orifice. If the regurgitant murmur is not heard well in the interscapular region and is not conducted down the vertebræ at all, the prognosis is good, no matter how loud it may be at the apex. But if it is heard all down the spine to the lumbar region, the damage to the valves is considerable and the outlook is more grave. Simple loudness is of much less serious import than conduction. Aortic disease is more serious than mitral and may prove suddenly fatal. Few cases live beyond puberty.

Endocardial murmurs due to anæmia or dilatation will disappear. That of true endocarditis may slowly disappear, and possibly remain permanently absent or reappear under strain in later life. Temporary disappearance during convalescence, the murmur returning when the child gets up, is of dubious outlook if it is truly valvular in origin and not functional.

The amount of hypertrophy, with or without dilatation, is the best measure of the magnitude of the lesion, provided the duration is taken into consideration. It is the proof that compensation has been more or less completely attained. If the hypertrophy is very great, it is not very likely to be lasting, for the more a muscle exceeds its normal bulk, the more difficult is it to maintain its normal nutrition and functional activity. Dilatation, if it has existed for some time and does not improve at once under treatment, is a proof that compensation has broken down and may not be regained. The effect on the circulation, arterial and venous, and the consequent changes in nutrition of distant organs and the body generally, and the presence of dropsical effusions, must also be considered. General improvement in nutrition and gain in weight are good signs. Malnutrition is a fair measure of the incapacity of the heart for its proper functions.

Compensation must be maintained as well as produced, so we must further consider whether the personal and social conditions of the patient are favourable to healthy cardiac nutrition. Favourable features are youth, a family tendency to longevity, equable temperament, strong will, and the absence of hereditary tendency to disease. The occupation and mode of life should be such that the patient can regulate at will the amount of work and rest, changes of climate, diet, and freedom from worry and excitement. Out-of-door exercise is of the utmost importance as long as it does not



cause pain, palpitation, dyspnœa, anorexia and sleeplessness. These children must not be brought up as chronic invalids.

The prognosis is particularly bad if a temperature persistently hectic in type or irregular and an enlarged spleen indicate infective endocarditis ; and if there are frequent severe attacks of cardiac pain.

*Treatment.*—The subjective symptoms of heart disease are constantly absent. When present they are commonly due to cardiac failure and include pallor, headache, dyspepsia, high colour, blueness of the extremities, dyspnœa, palpitations, pulmonary catarrh and so on, according to the nature of the mischief. The child is often indolent, irritable and capricious. In almost all cases the condition of the myocardium is the main indication for treatment, because of its great liability to dilatation during acute carditis and for a considerable time afterwards. Therefore, prolonged rest is the first and most cardinal measure in treatment. It should be continued for 1-3 months after acute symptoms have subsided and compensation has been established. As a rule children are allowed up much too soon. Special care is needed at puberty on account of the extra strain thrown on the heart by growth and development. The chief factors to estimate in the application of remedial measures are the nutrition and functional power of the heart muscle, the nature of the mischief and the valves affected, the idiosyncrasy and environment of the child, the irresponsibility of childhood, and the difficulty in securing the co-operation of the parents as soon as the troublesome symptoms have subsided.

Accurate diagnosis is of supreme importance. A murmur is not always a sign of valvular disease and some of the worst forms of heart disease have no murmur. Nor is digitalis always required when a murmur is present. Do not give heart stimulants, if recumbency alone is sufficient or after compensation is established. Use it carefully in small doses, for large ones may exhaust and injure a dilated feeble heart. Digitalis and strophanthus should be tried alternately to find out which suits the child best. Strophanthus is often the more efficacious in stenosis and bad dilatation. Strychnia is a useful adjuvant but cannot be relied on alone. In mitral stenosis digitalis is often beneficial, if given for a few days at a time. It should not be given in aortic regurgitation, except for failure of compensation and then the child must be kept in bed under strict observation. Drugs are most efficacious if engorgement of the right side of the heart is first relieved by dry cupping, leeches or venesection. As soon as compensation is fairly well established, give iron tonics.

Subsequently rely on good diet and hygiene to maintain cardiac and general nutrition. Regulate the amount and character of the exercise. In any case in which the myocardium has been affected or the endocarditis well marked, only allow walking exercise on level ground for 3-12 months ; and after this, minor gymnastics, hill climbing and bicycling on the level. Running, swimming, and prolonged athletic or competitive games must be



forbidden. Some of these children may be allowed to go to school, but such permission must only be given after the most careful consideration of all the factors in the case. Tea, coffee, alcohol and tobacco are contraindicated. Nervous excitement must be guarded against. If good compensation has been maintained for some years, the child can live pretty much the same life as its healthy comrades, especially if the lesion is only a mild degree of mitral regurgitation. It must be carefully watched for signs of cardiac strain, viz., anorexia, impaired nutrition, anæmia, dyspepsia, cough and shortness of breath.

Cardiac failure, due to weakness of the muscle, requires rest and tonic treatment. If it is associated with backward pressure, enlargement of the liver and congestion of the lungs, increase the power of the heart by strychnia, nux vomica, digitalis, squills, strophanthus, convallaria or caffeine. Mercury is most beneficial for the relief of congestion, as a diuretic and a mild purgative. Strong purgatives and diaphoretics are too severe remedies and only of temporary benefit.

Diuretin grs. 2-10, four times daily, sometimes succeeds when digitalis fails. One of the first signs of the benefit of digitalis is an increase in the secretion of urine. The amount should be measured daily and a marked increase is generally found in 3-4 days.

Mercurials, diuretics, purgatives, oxygen inhalations and, possibly, bleeding are indicated if the blood is de-oxygenated. Subcutaneous injections of strychnia are useful in cardiac fatigue, with sleeplessness and flatulent distension. The diet must consist of small easily digestible feeds. Limit the amount of fluids. Guard against over-feeding and setting up distension of the stomach. Alcohol, in small frequent doses, is needed in acute dilatation, myocardial affections and cardiac failure. Sal volatile is a valuable emergency stimulant. Morphia or codeia are perhaps required for severe dyspnœa, cough and insomnia.

**Pericarditis.**—Simple, plastic or “dry” pericarditis, localised or affecting the whole pericardium; pericarditis with effusion of serum, seropus, pus, fibrin or blood; hydropericardium and adherent pericardium occur in children in the same forms and from the same causes as in adults. They are due to direct extension from the lungs or pleura, thoracic glands, myocardium, thoracic walls, œsophagus, thymus or inflammation below the diaphragm; and to blood states or microbial infections, e.g., rheumatic fever, pyæmia, osteomyelitis, scarlet fever, nephritis, septic throat, otitis media, etc.

Rheumatism is the great cause and for this reason rheumatic pericarditis is rare before the seventh year, and most common from this up to the tenth year. Pericarditis in the newborn is due to puerperal fever or other infective disease in the mother, umbilical infection or vaccination. It is more often chronic than acute, frequently associated with pleurisy, and



usually fatal. Purulent pericarditis is set up by streptococci, staphylococci, the pneumococcus or the bacillus coli. The tuberculous form has been described above (p. 487). Pericardial effusion associated with pneumonia or empyema is nearly always purulent. A primary pneumococcal form may occur. Poynton (1908) analysed 100 fatal cases from various sources. Of these 83 were under 4 years of age; two-thirds from 1-3 years old. One possibly was primary. Empyema accounted for 60 per cent., either by direct extension, or a secondary or simultaneous blood infection; and 40 per cent. were associated with acute pleurisy or pneumonia. The frequency of pneumococcal pericarditis under 4 years of age is a striking contrast to the rarity of the rheumatic variety, and is to be expected on account of the susceptibility to pneumococcal infection in early life.

Anatomically there are found a few flakes of fibrino-pus and a little turbid fluid; a fibrino-plastic exudation, much as is seen in the rheumatic affection; creamy pus; or a thickened pericardium and inspissated pus. In a girl, aged 11 years, the pericardium was thick, velvety, and contained half a pint of thin streptococcal pus. Pleural adhesions were present at the left base and over the whole right lung. There was no endocarditis and no local source of infection discoverable. Her illness lasted 14 days and was characterised by high fever, a little swelling of the right knee and left ankle, sordes, dilated pupils, dyspnoea, delirium, hallucinations of vision and semi-consciousness. A more typical case was that of a boy, 21 months old, who was admitted with general bronchitis, cough and "bad breathing" of 2 weeks duration. The chief symptoms were attacks of collapse and lividity, subnormal temperature towards the end, very bad colour, frequent vomiting and an imperceptible pulse. The pericardium was covered with a thick, pile-like deposit of lymph and contained much sero-pus. Two small collections of greenish pus were present in the pleural cavity on the left side.

*Physical Signs.*—The signs of dry pericarditis are often limited to friction, which may be felt. It is called "*Latent*," when only found by chance on examination, rarely so in children. Friction varies in degree, character and extent. Its maximum intensity is at the base of the heart in the third and fourth interspaces, sometimes in the second, on the left side near the sternum. Occasionally it is limited to the apex and may simulate a presystolic murmur. It is localised over the place of production. If the inflammation starts behind, friction is not heard until it has spread anteriorly. The spread is often very rapid. At first the sound may be systolic. More generally it has a to-and-fro character like a double aortic murmur. It is increased by the pressure of the stethoscope, altered by change of posture, louder on sitting up, sounds much nearer the ear than an aortic murmur, and is not conducted beyond the cardiac area. Sometimes there is a single or double thrill. If the heart muscle is involved, signs of cardiac insufficiency develop.



Fluid is often present as well, sometimes localised. Large effusions are uncommon in rheumatic fever. As the fluid increases in quantity the physical signs alter. The precordium bulges. The apex is elevated and perhaps not palpable. It may alter with change in position and be only palpable in the genu-pectoral posture. The impulse is diffused, wavy and indistinct. Friction may disappear, through separation of the two layers of the pericardium by the fluid ; or it may be increased in the supine posture, through the heart being floated up more in contact with the chest wall. The left side of the chest is enlarged and its respiratory movements deficient or absent.

In small effusions there may be no change in the area of cardiac dulness. Dulness in the fifth right interspace is often the first sign, for in early stages the fluid collects in the lower part of the sac, especially at the right edge of the sternum. The cardio-hepatic angle, normally a right angle, becomes obtuse through displacement of the lung. In large effusions the shape of the dulness is that of a trapezium with the base downwards. It extends to the second rib and the fourth and fifth right interspaces, rendering the cardio-hepatic angle still more obtuse and the right border of the heart convex. It extends below and to the left of the apex and of the left edge of the heart. Difficulty arises in apportioning its proper share in the dulness to the dilatation of the heart so often present. There is an abrupt transition from the dull area to the resonant lung. Marked resistance is noted on percussion.

The heart sounds are progressively muffled, weak and distant, embryocardial in type, pendulum-like ; and there is often a murmur indicative of endocarditis. Compression of the lower lobe of the left lung produces a patch of dulness, somewhat quadrilateral in shape, and bronchial breathing or absent breath sounds near the angle of the left scapula. It may be mistaken for consolidation. Occasionally Skodäic resonance is present over the upper part of the lung.

*Symptoms.*—The onset is insidious, especially in the rheumatic affection. Arrhythmia, undue frequency of the pulse, slight fever, rather an increased rate of breathing, precordial pain and palpitations may be present ; perhaps only dyspnœa and slight fever ; or merely friction, limited in extent and found by chance on examination. A few cases show intense pain, urgent dyspnœa, high fever, sleeplessness and delirium. If there is a severe attack with considerable effusion, we find an anxious look, dusky pallor, dilating alæ nasi, and dyspnœa or orthopnœa, a most important sign and out of all proportion to the state of the lungs, temperature and pulse rate. It is partly due to pain, partly to congestion of the lungs. It may be so severe that the patient assumes weird positions, such as the genu-pectoral, and is only able to sleep in that posture. Cough is short, dry, frequent and distressing. One patient exhibited constant yawning and others have shown this to a less extent. Pain is moderate, occasionally



severe, and localised over the cardiac area if due to friction. It may be induced or increased by precordial or epigastric pressure, which may bring on nausea or vomiting. It often decreases as the effusion increases, and is at times due to acute dilatation. The large veins in the neck may be much distended. The pulse is very frequent, up to 180, quick, small and of low tension, perhaps very irregular and almost running. A *pulsus paradoxus* is rare, except in mediastino-pericarditis. Moderate fever, perhaps aphonia and dysphagia, and the usual signs of general malaise are present. Delirium is fairly common in rheumatic cases with hyperpyrexia. Acute mania has been noted.

A *purulent effusion* is suggested by symptoms unusual in the course of acute lung disease, pleurisy or empyema. Its course is acute, subacute or chronic, varying with the nature of the illness. It may come on quite insidiously during the convalescent stage of pneumonia. The chief signs are serious illness, lividity or livid pallor, panting, dyspnoea, orthopnoea, very frequent pulse and syncopal attacks, and irregular attacks of fever with feeble heart action. The temperature is not characteristic. It is generally irregular, with sudden falls below normal and attacks of collapse. The pulse rate is often 150-180, out of proportion to the discomfort and distress, and varies with the rate of breathing. Flabbiness, wasting, cyanosis, and oedema of the legs and feet may be present. Friction is usually absent. At times there is neither cardiac nor respiratory distress, and the signs may be limited to increased cardiac dulness and decreased loudness of heart sounds. The heart sounds are not always enfeebled. Nor is cardiac dulness invariably increased for the amount of effusion may be small, and is rarely large in young children. In small effusions the apex may be normally situated. In chronic cases there may be merely wasting and asthenia; sometimes vomiting and diarrhoea; or cough, pleural pain and wasting. In young infants a small purulent effusion may only be suggested by severe cardiac weakness of no apparent causation.

*Diagnosis.*—Increased dulness suggests effusion or dilatation. The chief difficulty is in differentiating acute dilatation from effusion, especially when they are conjoined or if the dilatation is associated with pleural effusion, with or without pneumonia. The position of the apex beat is a most valuable diagnostic point. Orthopnoea, weak apex beat, feeble sounds and increased dulness are the chief signs of effusion. Peritonitis may be suspected, if the pain is referred to the abdomen. Mediastinal tumours and pneumonia are occasional sources of difficulty. The nature of the fluid can be definitely settled by exploration. Hæmorrhagic effusions are due to purpura, scurvy and other blood affections, and to tuberculosis. Under 4 years of age the effusion is likely to be purulent because of the liability to pneumococcal infection, pneumonia, empyema and pyæmia. Later in life it is more apt to be fibrinous or sero-fibrinous. A scanty purulent effusion may give rise to no special signs or symptoms. It should always



be suspected in a child dying from empyema. A large one may be so extensive as to simulate empyema and be opened under that impression. Pus is also indicated by the serious general condition of the patient, attacks of lividity or collapse, and the presence of some source of pyæmic infection. In the absence of hyperpyrexia, the occurrence of delirium and maniacal symptoms is to my mind suggestive of pus; and other signs are progressive anæmia, wasting, and acute tenderness over the whole lower end of the sternum. In spite of these indications, it is by no means uncommon for the existence of pyo-pericardium to be only recognised at the autopsy. On account of the absence of a murmur attention is not drawn to the heart; and the presence of serious disease explaining the illness leads to the effusion being overlooked. Bronchitis and patches of purulent pleurisy may be mistaken for broncho-pneumonia, although pyo-pericardium is present. Its occurrence in marasmic infants is frequently ascribed to tuberculosis.

*Prognosis.*—A simple rheumatic pericarditis is not often fatal, for it rarely leads to copious effusion in children. The prognosis is serious for complete recovery is rare. It is often associated with endocarditis, or myocarditis which may end fatally; or results in adhesions, in which case the prognosis is that of adherent pericardium. The frequent pulse and respiration, anæmia, wasting and vomiting are bad signs, due to myocarditis rather than pericarditis. Purulent pericarditis is almost invariably fatal, sometimes from empyema or meningitis. A few cases have recovered after treatment by incision and drainage.

*Treatment.*—In dry pericarditis and rheumatic infection salicylates are given. Small doses of tr. opii, quartis horis, relieve the excited action of the heart. Apply to the precordium cold, mesotan, methyl-salicylate, lin. iodi, turpentine stupes or a mustard leaf. Small flying blisters and even leeches are occasionally permissible in older children. The production of blisters or local soreness interferes with examination of the heart. General treatment consists of absolute rest in the recumbent posture, avoidance of all exertion and excitement, light diet, and stimulants if there is much interference with the heart's action. Stimulants, such as alcohol, strychnia and ether, are necessary in effusion. Avoid digitalis for it prolongs diastole, permits dilatation of the cavities, and adds to the difficulties of a heart already hampered in its action by the mechanical effects of effusion. Diuretics and purgatives are of little value in its relief. The question of aspiration or drainage arises if there is much fluid. It is rarely necessary in rheumatic pericarditis, though it is probable that it could be used more freely with advantage. The indications for operation are a large effusion, marked cyanosis, great dyspnœa and a frequent, weak, irregular pulse.

Exploration must not be undertaken lightly. It is quite easy to wound the heart, an injury recognised at once by the movement of the needle.



Fortunately this is not a serious blunder. In fact the ventricle has been aspirated with distinct advantage in dilatation. A slight injury to the ventricle is soon closed, owing to the peculiar arrangement of the muscular fibres. A wound of the auricle may cause uncontrollable hæmorrhage and death from hæmo-pericardium. Sudden death has also resulted from inhibition. Other risks are the wounding of a large vessel and infection of the pleura by septic material from the pericardium. Simple paracentesis may prove negative from a diagnostic point of view, although a small amount of pus is present.

It is sometimes advised that the exploring needle should not be used and that incision and resection must be adopted even for diagnosis. This is a counsel of timidity. With reasonable care exploration is safe and satisfactory, and can be used in circumstances which would not justify recourse to the more serious operation. For exploration or aspiration only a local anæsthetic is needed ; for intercostal incision, resection and drainage a general one is required. Use a small needle  $1\frac{1}{2}$  in. long, attached to a rubber tube and syringe. The safest place for its insertion is between the apex and the extreme limit of dulness in the axilla (S. West) although the pleura is punctured. Avoid the site of friction. Push the needle in slowly and stop as soon as the heart is felt. Drain off fluid slowly by syphonage, attaching a larger tube.

In choosing the site of operation bear in mind the position of the heart, for it may float up in approximation with the chest wall. Consider too the position of the internal mammary artery and that the relations of the right pleura to the thoracic walls are not altered by pericardial effusion. It extends well to the middle of the sternum, perhaps to the left edge. The site depends partly on the physical signs. Some surgeons recommend the fourth or fifth left space, as close to the sternum as possible, trying the fifth first, and if both fail then trying  $1\frac{1}{2}$  in. from the left edge of the sternum ; or the fourth and fifth right spaces, although the pleura is certain to be punctured ; or the sixth space in the nipple line below the apex if it is palpable.

The site of *incision* should be chosen with a view to securing efficient drainage at the time and when the sac contracts. There is no serious objection to draining through the pleural cavity, for the pericardium rapidly becomes adherent after the evacuation. Cases opened as empyemata have done well. The incision should extend from the middle of the sternum over the fifth left costal cartilage to its junction with the rib ; the cartilage is excised, and the internal mammary vessels ligatured in two places and divided ; the fibres of the triangularis sterni are separated, and the pleura identified and pushed aside ; the pericardium exposed, and an incision made downward and outward from the sternum ; the edges of the incision are stitched to the soft parts. Or a vertical incision is made and the sixth cartilage, perhaps the seventh also or the seventh and part of the gladiolus,



is removed. Others recommend an incision in the fourth left space, beginning 1 in. from the sternum and reaching to the apex. West advises incision in the apex region and no resection ; and probably this is the safest and best mode of operation. A large rubber tube is inserted. The cavity is irrigated daily with warm fluid, provided there is a free exit for the fluid, and the patient is turned on the side or face, if not too ill, to facilitate drainage. The tube is removed in 3-4 days if the effusion is serous. The mortality is about 60 per cent. It depends on the cause ; is least in pneumococcal cases and highest in pyæmic ones, even though the pericardium becomes adherent a few days after the operation. Aspiration is much more dangerous than open incision.

**Mediastino-pericarditis** was described by Griesinger in 1854. Cases have been reported under the names of indurative mediastino-pericarditis, obliterative pericarditis with ascites, pericarditic pseudo-cirrhosis of the liver, polyserositis, and “ zuckergussleber ” (Curschmann). It is a chronic inflammation of the pericardium and mediastinum, insidious in onset, and septic, rheumatic or tuberculous in origin. Tuberculosis of the pericardium is described above (p. 487). The inflammation may spread from the glands to the mediastinum and thence to the pericardium, or be primary in the pericardium and not necessarily followed by mediastinitis. Tuberculous cases have been reported at the age of 15 months (Sequeira) and 2 years (Ashby). Others have followed specific fevers, notably measles, and rheumatic pericarditis.

The pericardium may be merely adherent. More generally it is very greatly thickened, fibrous or almost cartilaginous. In a girl, aged 13, it contained large calcareous deposits (Bennion). It is universally adherent to the heart, sometimes irregularly adherent with saccules of purulent fluid ; adherent to both layers of the pleura and the sternum ; and the inflammation spreads upward around the great vessels. The anterior and posterior mediastinal tissues are much thickened and adherent to the pericardium. The fibrous tissue slowly contracts, interferes with the growth of the heart, and leads to mechanical obstruction to the return of blood to the heart, enlargement of the liver, ascites, anasarca of the legs and cyanosis. The other serous membranes, especially the peritoneal covering of the liver, may be involved. The pathological changes in the liver, spleen and kidneys are those of venous engorgement. The ventricles are dilated and hypertrophied, and the valves usually healthy.

Cough, shortness of breath, giddiness and debility may be the first symptoms. In early stages the child is pale but looks healthy, has a clean tongue, and no undue frequency of pulse or breathing. Later on it develops swelling of the face and legs, coldness and blueness of the extremities, persistent cyanosis which is marked in the lips and ears, distension of the veins in the neck, much enlargement of the liver and perhaps of the spleen, increasing ascites and œdema of the abdominal wall. Inspiratory collapse.



of the veins in the neck is a noticeable sign. The pulse is frequent, and a pulsus paradoxus is more common in this than any other disease, but may be present in acute pericarditis and even in health. The symptoms are chiefly those of cardiac dilatation or abdominal in character. Alternating attacks of diarrhœa and constipation, abdominal pain, and pleurisy are not uncommon. Dyspnœa is marked. Albuminuria is usually absent, except in late stages. Ascites may appear before the anasarca. The heart is not always enlarged and generally presents no adventitious sounds. The signs of enlargement may be present and extend upward along the great vessels, to the level of the second rib of the left side.

The course is chronic. Bosanquet's 3 patients, boys aged 6, 8 and 15 years, lived 2-3 years. The usual duration is 2-6 months. Death results from asthenia, diarrhœa, broncho-pneumonia or other complication. The outlook is most hopeful in cases of rheumatic origin.

It is liable to be mistaken for tuberculous peritonitis or cirrhosis of the liver. Stress must be laid on the dilated veins and their inspiratory collapse, pulsus paradoxus, œdema and ascites, and the absence of valvular disease. Hepatic enlargement with persistent pleural effusion in children, without obvious cause, is suggestive. The large, tense, tender liver is out of proportion to the venous stasis. Treatment is directed to symptoms. Possibly potassium iodide may do good. Paracentesis may be needed for the relief of ascites or pleural effusion.

**Adherent Pericardium.**—The simple form of adherent pericardium, commonly of rheumatic origin, must be distinguished from the preceding condition of mediastino-pericarditis, though there is no strict line of demarcation. It is often associated with adhesions to the chest wall and diaphragm, perhaps pleurisy and mediastinitis. It has been found in the newborn and at 3 months of age, but is rare before 7 years. It is liable to occur whenever there has been pericardial friction heard in the course of illness.

It must be suspected in cases of marked cardiac hypertrophy, when the valvular mischief does not seem sufficient to account for the degree of enlargement; in the presence of ascites and anasarca, associated with a valvular lesion, when the cardiac impulse is forcible and the heart sounds seem good; and in cases of "quiet" ascites, in which hepatic cirrhosis, tuberculous peritonitis and malaria can be excluded. Except the first, these conditions are rather those of mediastino-pericarditis than simple adherent pericardium. Hypertrophy due to chronic interstitial nephritis must not be forgotten.

The orthodox signs are often conspicuous by their absence, and the condition may be quite unsuspected and only found after death. It is on account of the difficulty in excluding mediastino-pericarditis that differential diagnosis between the two affections is so great. One of the most valuable signs is an abnormally frequent pulse. If it remains very frequent in spite of treatment, more frequent than any cardiac murmur present will



account for, and particularly if there is no great hypertrophy, it is almost certain that the affection is present. It is one of the chief causes of hypertrophy and yet the large heart only produces a frequent, persistently feeble pulse, and the apex beat may be imperceptible. Epistaxis is said by Letulle to be a symptom.

The diagnosis is based on a combination of some of the following physical signs:—hypertrophy of the heart, weak heart action, weak and frequent pulse, marked undulation of the heart beat, imperceptible apex beat, absence of postural changes in the apex beat, absent impulse without increase in the area of dulness, and the usual signs of cardiac insufficiency. Hypertrophy may be absent, if the pericardium is universally adherent. If the pericardium is adherent to the chest wall there is systolic retraction of the intercostal spaces, and perhaps the ribs, in the region of the apex, and even of the entire precordium. Systolic retraction, limited to the apex, is of no value for it occurs in other affections. There is no change in the area of dulness on inspiration or change of posture, for the edge of the lung on expansion cannot extend between the heart and chest wall. Retraction of the lower portion of the left chest in the lateral and posterior aspects, absence of respiratory movements in the epigastric triangle, and a diastolic shock and rebound may be present, if there is mediastinitis and adhesions of the pericardium to the chest wall and diaphragm, for the diaphragm cannot descend properly. A presystolic murmur has been noted in some cases, in the absence of valvular mischief, and is ascribed to cardiac dilatation. Other signs, such as cyanosis of lips and ears, hepatic enlargement and ascites, are also evidence of mediastino-pericarditis. Thus it is obvious how difficult is the differential diagnosis of the two affections, and that many of the signs commonly ascribed to adherent pericardium are those of the more extensive disease.

It is by no means invariable for pericarditis to be followed by adherent pericardium, at any rate not to an extent which interferes with growth and development. I have known a most prolonged and serious case apparently clear up entirely, and the girl, 6 years later, aged 15 and well grown, showed no sign of cardiac mischief. Good nutrition and growth are favourable signs. Nevertheless the outlook is most grave and few patients reach adult life, dying from cardiac failure and asthenia, sometimes suddenly. It must be regarded as a more serious sequence of rheumatism than valvular disease.

*Treatment* is entirely symptomatic, and that of cardiac failure on the lines described in the general treatment of heart disease (p. 493). Resection of rib cartilages in the regio cordis has been recommended, if there is strong retraction of the ribs indicative of extensive adhesion to the chest wall. It lightens the work of the heart, but, even if successful, is unlikely to be of more than temporary benefit.



## CHAPTER XLIII.

### THE BLOOD AND LYMPHOID STRUCTURES.

*The Blood—Primary Anæmias—The Spleen—Anæmia Splenica Infantum—Splenic Anæmia—Leukæmia—Chloroma—Glandular Affections—Glandular Fever—Lymphosarcoma—Lymphadenoma—The Thymus—The Status Lymphaticus.*

**Examination of the Blood.**—To obtain a sample, prick the lower surface of the lobule of the ear with a sterile triangular needle. This area is very insensitive and can be pricked without waking a sleeping child. Wipe away the first few drops and do not use blood obtained by squeezing. Take the sample before a meal, to avoid error from the leucocytosis of digestion, which is well marked in infants and children. Make blood-films by pressing a minute portion between two cover-slips, separating them by a sliding movement, and dry them slowly over a spirit flame. To make a fresh preparation, take up some staining fluid, 0·2 per cent. methylene blue (Grübler) in 40 per cent. alcohol, in a Thoma-Zeiss pipette. Mix it gently with a largish drop of blood on a glass slide, by blowing the staining fluid on to the blood. The colour turns deep green. Put on a cover-slip and seal the edges with vaseline. In this method the white cells are undistorted by pressure. In addition, make a fresh preparation by taking a minute portion of blood on a cover-slip and inverting it gently on the slide. Examine this at once for blood-plates, rouleaux formation and fibrin strands. A fair idea of the degree of anæmia, poikilocytosis and leucocytosis is at once obtained.

The *red cells* are counted by means of a Thoma-Zeiss or Gower's hæmatocytometer. In examining for poikilocytes fix the red cells first by osmic acid solution 1 per cent., containing 0·2 per cent. methylene green. Or use as a diluting fluid, Toison's solution (methyl violet 0·025, sod. chlor. 1·0, sod. sulphat. 8·0, neutral glycerine 30·0, distilled water 160 parts), filtered before use. Another good diluting fluid is Edington's, consisting of neutral sod. citrat. 7·5, formalin (40 per cent.) 2·0, Grübler's dahlia 0·03, chloroform m5, water 250 parts. Count 5 fields of 36 squares each, repeat the process with three different drops of the diluted blood, and then take the average of the two results which most approximate. For counting white cells dilute the blood with glacial acetic acid solution, 0·33 per cent. strength, Toison's or Edington's fluid, or methylene green or blue



0·2-0·4 per cent. in 40 per cent. alcohol. Clean the pipette with distilled water, sulphuric acid, distilled water, ether, absolute alcohol and hot air successively.

For *differential staining* use Ehrlich-Biondi fluid, Ehrlich's triple stain or acid dahlia solution, Jenner's stain, or eosin and methylene blue, either separately or in the form of Canon's dye. To estimate percentages count 1000 cells.

Von Fleischl's or Gower's hæmoglobinometer is used for estimating the *percentage of hæmoglobin*. They depend on colorimetric methods and are not very reliable. The *specific gravity* gives a fair estimate. It is obtained by letting a drop of blood fall into a mixture of benzol and chloroform of specific gravity 1059, that of normal blood. If it sinks add more chloroform, if it rises to the surface add benzol. When the drop floats like a bead in the mixture, the specific gravity of the mixture is the same as that of the blood. The lower the specific gravity, the lower is the percentage of hæmoglobin, and *vice versa*. The *colour-index* is represented by a fraction, with numerator the percentage of hæmoglobin and denominator the percentage of red cells. The red cells are erythrocytes, macrocytes, microcytes or poikilocytes according as they are normal, larger than normal, smaller than normal, or irregular in shape. Nucleated red cells or hæmatoblasts are divisible according to size into normoblasts, megaloblasts and microblasts. Megaloblasts are found in foetal marrow and grave forms of anæmia. Microblasts are rarer. Normoblasts resemble ordinary erythrocytes, except for containing a nucleus. They are present in marrow in small numbers; after hæmorrhage, in large numbers. They are usually regarded as immature erythrocytes.

The *white corpuscles* are non-granular or granular. Their protoplasm does not stain with methylene green. The non-granular cells, usually known as *Lymphocytes* or mononuclears, stain with basic dyes. They are usually regarded as the youngest form of leucocyte and are divided into:—(1) *Small lymphocytes*, consisting of a round nucleus about the size of an erythrocyte, surrounded by a narrow zone of faintly staining protoplasm. The smaller the nucleus, the more deeply it stains. They are said to grow by increase in protoplasm and nucleus into the next variety. (2) *Large lymphocytes* or *large hyaline cells*, about twice the size of the small type and much paler. The nucleus is large, stains feebly, and is surrounded by a bigger zone of protoplasm. (3) *Transitional or intermediate forms* occupy a position between the other two. Some are large and oval. Some have an indented or horseshoe-shaped nucleus.

The granular cells include:—(1) *Polymorphonuclear cells*, sometimes called polymorphs or polynuclears, neutrophiles, or finely granular oxyphiles. The granules stain faintly with acid and neutral dyes, the latter being really faintly acid. They are regarded as the full-grown type of leucocyte. They are intermediate in size between a small and large



lymphocyte, and contain many fine granules and an irregular or multipartite nucleus, situated in the centre of the cell and staining deeply but unevenly with basic dyes. The granules surround the nucleus, and neither the protoplasm nor the granules stain with methylene blue. (2) *Eosinophiles*, cells resembling the last variety except in the granules. They are coarsely granular, oxyphile cells, regarded as old or overripe leucocytes. The granules are large and stain with Ehrlich-Biondi fluid, eosin, acid fuchsin and other acid dyes, and greenish with methylene blue. The protoplasm is stained by the last dye. Eosinophilia is found in infants, bronchitic asthma, some skin diseases, and at times in leukæmia, bone disease, worms, gonorrhœa and at puberty in girls. (3) *Mast-cells* are basophilic and resemble eosinophiles but are much smaller, about the size of small lymphocytes. The granules are large and not numerous. They stain deeply with methylene blue, but in specimens stained with Ehrlich-Biondi fluid they appear as clear white spots. The protoplasm stains mauve or purple with methylene blue. These cells are few in number or absent. (4) *Myelocytes or marrow cells* can be recognised on staining with Ehrlich-Biondi fluid. They are like large lymphocytes but contain a great many granules; and the nucleus almost fills and is in close contact with the wall of the cell, being large, spherical or egg-shaped, and eccentric, instead of centrally placed. On staining with Jenner's dye they are subdivisible into neutrophilic or ordinary myelocytes, the granules of which stain blue; and a few eosinophilic ones, the granules of which stain red. The relative proportion does not appear to have any special significance. The granules are small, as in the polymorphs. These cells possess no amœboid movement. They are supposed to be an intermediate stage between the large lymphocytes and the polymorphs, though they are usually larger than both varieties.

Myelocytes, many eosinophilic, are normally present in the marrow. They are occasionally found in the blood of the newborn and are rarely entirely absent during the first few weeks of life. In various conditions of anæmia and splenic enlargement they are found in quantities from 1-20 per cent. of the total number of white cells. They are often present in congenital syphilis, rickets, tuberculosis, pneumonia and entero-colitis. Their presence is due to the introduction of toxins, and it is doubtful whether they have any diagnostic or prognostic significance. They are apt to be more abundant in the younger infants on account of the tendency of the blood to revert to the more infantile type in the course of illness.

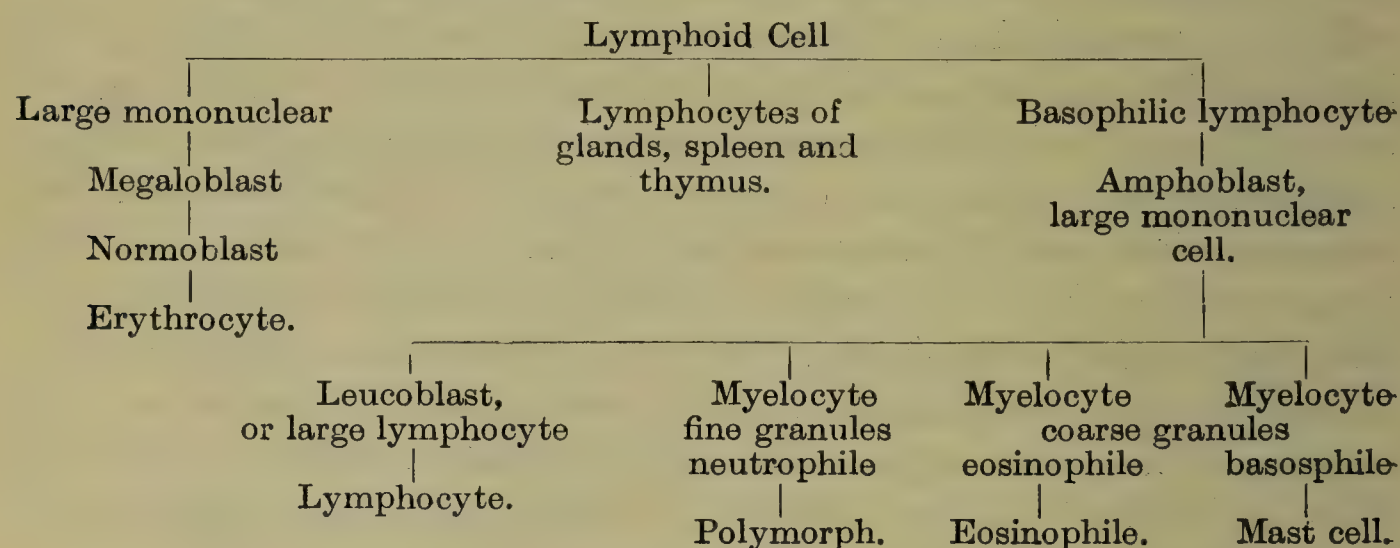
The *reaction of the blood* is valueless for clinical purposes at present. It is practically neutral, and not alkaline in the sense that sodium hydrate is alkaline. The salts are mainly carbonates and phosphates of the alkalies. The bicarbonate and phosphate of soda are really acid salts with an alkaline reaction to many reagents. It is extremely difficult to make the blood either more or less alkaline.



*The Origin of Blood Cells.*—Up to a certain stage of embryonic life the blood cells are developed from a primitive mesoblastic cell, an original “indifferent” cell of a lymphoid type with a large oval nucleus and basophile non-granular protoplasm. According to a classification by Treadgold (1908) three varieties of cells are derived from the primitive one.

The first passes through the stages of megaloblast and normoblast into the red cell or erythrocyte. The second develops eventually into the lymphocytes of lymph glands, thymus and spleen. And from the third is formed the basophilic lymphocyte of the early embryonic circulation from which arises the large mononuclear cell (amphoblast). This gives birth to lymphocytes and myelocytes, and from the various types of myelocytes are developed the polymorphs, eosinophiles and mast cells. Treadgold’s classification may be tabulated in an abbreviated form as follows :—

*The Development of Blood Cells.*



Before birth the lymphadenoid and myeloid tissues become differentiated and the granular cells arise from the marrow, the non-granular cells from the lymphoid tissue. The marrow contains foci of lymphadenoid tissue, so some of the non-granular cells arise from the marrow though not from myeloid tissue. Possibly on a reversion to foetal conditions, or a more infantile blood formation, as the result of illness, the origin of the non-granular and granular cells may be reversed or take place in both ways. In infancy there is a great amount of lymphoid tissue and its activity is much increased during ill-health. Its function is doubtful. Possibly it is the chief agent for the destruction of toxins, while the micro-organisms are destroyed by cells which take their origin from the bone marrow.

The *bone marrow* contains two varieties of red cells, non-nucleated blood cells and nucleated red marrow cells. The white cells are non-granular and granular. Of the non-granular ones 50-70 per cent. are faintly basophilic, lymphoid or primitive cells, the size of erythrocytes and 1 per cent. are giant or mother cells, faintly neutrophilic and containing



many nuclei. The granular cells are the ordinary myelocytes, finely granular oxyphile or neutrophile (30-40 per cent.); the eosinophile myelocytes, coarsely granular oxyphile, the most constant and typical; basophile myelocytes, like the eosinophile ones except in the nature of the granules; and polymorphs from the blood (C. P. Jones, 1905).

*The Blood in the Newborn.*—The amount of blood is proportionally to the weight less than in the adult, unless the reserve blood is pressed out of the placenta before the cord is tied. The reaction is alkaline to litmus, and the colour rather darker during the first few days of life than later. The chief differences are best shown in the form of a table.

*The Characters of the Blood.*

	AT BIRTH.	AT 6 MONTHS.	ADULT.
Specific gravity .. ..	1062-66	1048-52	1058-62
Hæmoglobin .. ..	110	70	100
Red cells per c.mm. ..	5-6,000,000	4,500,000	5,000,000
White cells do. .. ..	20,000	15,000	10,000
<i>Percentages—</i>			
Small lymphocytes .. ..	} 16·05 {	50-70	20-30
Large lymphocytes .. ..		4-8	5-15
Polymorphonuclears .. ..	73·4	20-40	60-75
Eosinophiles .. ..	—	1-10	0·5-4
Mast cells .. ..	—	few or absent	up to 0·5

The differential count for leucocytes in the newborn is that obtained by Carstangen on the first day of life. The “leucocytosis of the newborn” is one of polynuclear cells. The number of these cells falls by the end of the first week to a level which remains fairly constant throughout life. After the first week there is a relative lymphocytosis. No adequate explanation of the leucocytosis of the newborn has been put forward. During the early days of life and throughout childhood the percentage varies greatly and the “leucocytosis of digestion” is marked. The adult type of blood is not fully attained until the fifth year, possibly not until the eighth.

In the third year of life the percentages of lymphocytes and polymorphs are about equal. A leucocytosis is normal in infancy but indicates disease in later life. Similarly, lymphocytosis is characteristic of infancy, but after 3 years of age it is abnormal. In all disease conditions the blood reverts to a more infantile type and the blood is like that of a younger child. The characters of the blood vary as the development of the child and causes, which retard development, have a retarding effect on the blood.



The percentage of hæmoglobin is high at birth and falls steadily for 6 months, then remains stationary up to 2 years of age, and slowly rises to the adult level in the sixth year. Sometimes it falls to the minimum level in the first few weeks of life, remains stationary until the sixth month and then slowly rises. It is generally lower in children than in adults, and higher in boys than girls. The red cells are in excess at birth and perhaps for a few days after. They diminish during the first few months and then increase to the normal level by about the eighth year. Microcytes are more numerous, the cells more spherical and with less tendency to rouleaux formation, and "shadow corpuscles" are more plentiful for the hæmoglobin is less closely combined with the protoplasm. Oxygen seems to be more firmly combined. Milne Murray found that it required seven times as much reducing agent to get a reduced spectrum. Normoblasts and megaloblasts are present in foetal blood, and are often found in considerable number on the first day of life, especially in the premature. They disappear by the end of the fourth day. Coagulation is incomplete. Scherenziss states that there is only one-third the amount of fibrinogen present compared to that in adult blood. There are more sodium and less potassium salts than in adults. The most important differences are the deficiency of hæmoglobin, a relative chlorosis, and the excess of lymphocytes. These cells vary more in size and many are larger than in adults. Leucocytosis and grave anæmia are produced by much more trivial causes.

*Leucocytosis* is of value for diagnostic purposes. It is normal in the newborn, after cold baths, massage and exercise, during digestion, and sometimes in the dying. It is present in gastro-enteritis, appendicitis, septic peritonitis, bronchitis, pneumonia, pleurisy, pericarditis, rheumatic fever, septic and cerebrospinal meningitis, advanced phthisis, scarlatina, variola, diphtheria, tonsillitis, whooping cough, erysipelas, osteomyelitis, septic and pyæmic conditions, rickets, leukæmic affections, and some diseases of the skin, liver and kidney. It is absent in early phthisis, miliary tuberculosis, tuberculous meningitis, enteric fever, measles, rōtheln, mumps, influenza (generally), malaria, and some varieties of pleurisy, pericarditis and peritonitis. Thus it is present in most septic and febrile disorders. It may also be induced by antipyrin, pilocarpin, salicylates, large doses of quinine, thyroid extract and tuberculin injections.

It is present in inflammation, if the intensity of the infection can excite reaction and if the patient's resistance is capable of reaction. Hence the degree of leucocytosis is of value in prognosis, provided that the toxæmia is not so intense as to prevent reaction. In febrile disorders it may occur before the fever, and bears no relation to its severity. In pneumonia a "blood crisis" may precede the temperature crisis.

*Leucopenia* may be found in some of the affections in which leucocytosis is absent, e.g., enteric fever, measles, influenza and uncomplicated tuberculosis; also in malnutrition and starvation.



**Anæmia.**—The classification of the various anæmias into primary, secondary and intermediate varieties is unsatisfactory and of no assistance in understanding the pathological changes. Anæmia is due to a multiplicity of causes. A few cases are congenital in origin, dependent on syphilis, tuberculosis, malaria, delicacy or general malnutrition in the mother. Usually it is symptomatic, a secondary result of improper feeding, a prolonged diet of cooked milk, insufficient protein or deficiency of iron containing foods; gastro-enteric derangements, tuberculosis, fevers, suppurations, renal disease, hæmorrhage and hæmorrhagic affections, such as purpura, scurvy and hæmophilia; and poisons, e.g., lead, mercury, chlorate of potash and coal tar products. Intestinal toxins can only cause hæmolysis and anæmia, if they escape destruction in the liver and pass into the blood. Their effect on lymphoid tissue is to produce lymphocytosis. On the marrow the effect is first an increased production of polymorphs and eosinophiles; and later the production of myelocytes, due to the failure of their conversion into more matured cells. The red cells are destroyed in the blood; hæmatoblasts appear; and the spleen becomes enlarged in order to dispose of the red cells and leucocytes. Probably other toxins act in a similar manner. There may be a deficiency of hæmoglobin, as in scurvy; destruction of red cells, as in diphtheria; or both, as in rheumatic and infective fevers.

Examination of the blood enables us to estimate the severity of the process. In mild cases the percentage of hæmoglobin is decreased and the blood assumes the chlorotic type. Its specific gravity is lowered. In more severe ones the red cells are reduced in number, and poikilocytes and hæmatoblasts appear in varying degree. This is a striking feature in early life. The field for blood formation cannot be increased in area and overstimulation leads to imperfection of its products. Leucocytosis may be present as the result of the primary or of coincident disease. The percentage of the different cells is unaltered or the polymorphs are in excess. Except in mild cases without leucocytosis the spleen is almost always enlarged. Put shortly, the greater the degree of anæmia the greater is the reduction in the specific gravity (down to 1035); the more the reduction in red cells (down to 2 million) and the percentage of hæmoglobin (down to 30 per cent.); the more marked the poikilocytosis and polychromatophilia; the greater the number of normoblasts and megaloblasts; and the greater the leucocytosis. A few myelocytes may be present.

The *symptoms* are pallor, fretfulness and irritability, shortness of breath on exertion, digestive troubles, constipation, headache, insomnia and muscular debility.

The *diagnosis* depends on the characteristics of the blood and the recognition of the cause. Splenic hyperplasia suggests leukæmia or anæmia splenica infantum, etc.



The *prognosis* varies with the cause, age, extent of blood changes, and general condition of the patient. Anæmia due to simple hæmorrhage is readily recovered from. In other cases it depends on the severity of the cause and is often that of the primary disease. In a few instances an apparently mild type develops into a fatal one. Death is usually due to intercurrent disease.

**Chlorosis**, sometimes called "oligosideræmia," is rare in early life and in breast-fed infants. The age of most recorded cases is from 11 months to 3 years. One infant, seen in consultation, was only 8 months old. He was born in the eighth month of pregnancy and only weighed 3 lb. 9 oz. at birth. He had been brought up on peptonised milk and then on boiled milk, cream and water. When seen, he was profoundly anæmic and weighed 14 lb. 6 oz. Six months later there was no anæmia. Another case was also one of twins, one month premature. At 9 months of age he had acute summer diarrhœa and at 16 months was profoundly anæmic. This affection is most often found in twins and premature infants fed on prolonged milk diet. According to Bunge reserve iron is stored up in the liver *in utero*, and it is stated that this accumulation takes place in the last three months of pregnancy. Thus a premature infant is likely to have an inefficient store and cannot obtain sufficient from a milk diet.

The complexion is a pale yellow, often greenish tint. The mucous membranes are pale. The child is puffy, soft, flaccid, under weight and costive. Sometimes there are diarrhœa and digestive troubles which yield readily to treatment. Apathy, mental dulness and hæmic murmurs are present. The spleen is not enlarged. The percentage of hæmoglobin falls to 50 and the blood shows poikilocytes and polychromatophilia, but is otherwise normal.

**Pernicious Anæmia** is almost unknown in childhood. Of 240 cases collected by Ehrlich only 1 was under 10 years of age. Some of the recorded cases will not bear strict investigation, and are really secondary anæmias. It is a primary anæmia which exhibits definite blood changes, progresses rapidly and almost invariably ends fatally. A fatal anæmia is not necessarily pernicious anæmia. In view of the theory that the disease is caused by septic infection *plus* a specific hæmolytic poison and that the infection is commonly buccal in origin, it is remarkable that more cases are not seen in children for they are particularly liable to mouth affections and almost constantly possess one or more carious teeth.

The blood is watery, dirty pink, reddish brown or coffee-coloured. It clots feebly and does not form rouleaux. The red cells fall to  $\frac{1}{2}$ -2 millions per c.mm., hæmoglobin to 15-25 per cent., and the specific gravity to 1030. The colour index is normal or high, never subnormal as in secondary anæmia. The other blood changes include poikilocytosis, polychromatophilia, a preponderance of megalocytes, microcytes, shadow corpuscles, megaloblasts and normoblasts, karyokinesis of the nuclei of red cells, perhaps a few



myelocytes, and a normal number or deficiency, rarely an excess, of white cells. A relative lymphocytosis has been found in bad cases.

Iron is deposited in the liver and intestinal mucosa, and is in excess in the kidneys and spleen. The spleen may exhibit some enlargement from fibrous hyperplasia. The bone marrow is embryonic in type, and contains many megaloblasts and normoblasts. Warthin states that the prevertebral lymph glands, especially the cervical, thoracic and retro-peritoneal ones, are affected.

The symptoms, course and prognosis are the same as in adults. An insidious onset is followed by profound anæmia and prostration, without emaciation. Headache, fainting attacks, cardiac dilatation, hæmic murmurs, œdema, and sometimes fever, are present. Anorexia, nausea and vomiting are common; diarrhœa not infrequent. The spleen may be palpable. Hæmorrhages take place from the nose, subcutaneously, into the retina, from the stomach, bowel, lungs, etc. The prognosis increases in gravity as the megaloblasts exceed the normoblasts in number. It ends fatally in a few months to a year.

**The Spleen.**—The spleen is one-tenth the weight of the liver at birth;  $\frac{1}{2}$ - $\frac{3}{4}$  oz., varying from  $\frac{1}{4}$ -2 ozs. It doubles its weight in a year, trebles it in 3-7 years, and quadruples it in 7-14 years. It is not essential to life and its functions are not clearly understood. Probably it acts as a filter for the removal of foreign particles from the blood, e.g., debris of red and white cells, and is consequently enlarged in diseases which lead to blood destruction. It is lymphogenic, producing non-granular lymphocytes, and perhaps contains indifferent cells which can develop according to the body needs.

It is much enlarged in myelogenic leukæmia, anæmia splenica infantum, infective endocarditis, malaria, and some cases of hepatic cirrhosis, typhoid fever and infarction. It is enlarged to a less extent in the early stages of these diseases and in lymphatic leukæmia, amyloid disease, congenital syphilis, rickets, portal obstruction and backward cardiac pressure. To a certain extent it is enlarged in all fevers. Occasionally the increase in size is due to abscess, hydatid, caseous masses, gummata or traumatism.

A moderate enlargement causes a little increase in the splenic area of dulness and a palpable lump, felt below the costal arch on inspiration. If the spleen is very large it causes bulging on the left side in the hypochondrium, and a distinct tumour, which moves on respiration, is close to the costal margin, and has a sharply defined edge with a notch. The inner edge may stand up as a well-marked ridge and the notch be visible through the thin abdominal wall. The percussion note is dull in front, except perhaps over the lower anterior edge if a coil of intestine happens to become interposed between the abdominal wall and the spleen. In the left loin it is resonant, for the tumour is covered by the descending colon.



It has to be diagnosed from renal and adrenal tumour and abscess, tuberculous peritonitis, masses of enlarged glands, fæces, ovarian cysts, pancreatic cysts and tumours, and malignant disease of the glands, splenic flexure, stomach or omentum.

**Anæmia Splenica Infantum.**—*Syn.*: *Anæmia Infantum Pseudo-leukæmica* (von Jaksch), *Anæmia splenica infettiva dei bambini*, *Splenic anæmia*.—In this affection anæmia and enlargement of the spleen are combined, without any characteristic changes in the blood. There is no definite evidence of a primary cause and yet there are sufficient grounds for looking upon it as a disease *sui generis*, rather than a secondary anæmia. Possibly it is due to some toxin which gives rise to both the anæmia and the splenic enlargement; the hyperplasia not being due to the anæmia, nor the anæmia a secondary effect of the hyperplasia, for both can occur independently. It affects both sexes equally, and is most common at 6 months to 2 years of age. Twins are particularly liable. Much argument has been devoted to its connection with rickets and syphilis. No doubt both these affections can cause splenic hyperplasia and possibly anæmia, but the disease under consideration is found sometimes quite independently of the least evidence of either affection. Rickets is not a cause of anæmia and the degree of rickets, when present, bears no proportionate relation to the blood changes or the size of the spleen. It is coincident, not causative. The same factors concerned in the production of splenic anæmia may induce rickets. In my experience there is rarely a family history or direct evidence of syphilis. The disease is more common among Jews, especially Polish Jews, than Gentiles. Yet syphilis is said to be less common among Jews and more of their children are breast fed. It is most frequent in hand-fed infants brought up on proprietary foods and condensed milk. Often there is a past history of alimentary disturbance and bad hygienic surroundings. Possibly these are concomitant factors or the primary cause is a toxin of intestinal origin. There is no doubt that many varieties of anæmia, usually secondary, in which the spleen is enlarged, have been described under this heading, but there is a definite residuum of cases which we are justified in regarding as a distinct entity.

**Morbid Anatomy.**—There are no special anatomical changes peculiar to this disease. The spleen is large, often enormous, and has a firm rounded edge. Its size bears no relation to the number of red cells or the degree of leucocytosis, if any. The enlargement is a simple hyperplasia without any leukæmic infiltration; and similar changes are found in both rickets and congenital syphilis. The degree of fibrosis depends on the duration of the disease. The liver is generally, but not always, larger than normal and its size in no way depends on that of the spleen. It may not be at all enlarged though the spleen is enormous. Microscopically it is normal, fatty, or may show foetal characteristics. In about half the cases there is some



hyperplasia of the lymph glands. No characteristic changes are found in the marrow and there are no leukæmic changes throughout the body.

*Symptoms.*—The onset and course are insidious, the pallor being the most striking feature. The complexion is waxy, with a tinge of olive green. Shortness of breath, dilatation of the heart and hæmic murmurs are present. Sometimes the enlarged spleen is present before the anæmia. The child often remains plump, though the anæmia and debility steadily increase. Occasionally there is marked emaciation. One boy, 8 months old, had dusky gums but no other signs suggestive of scurvy. The chief gastro-intestinal symptoms are anorexia, vomiting and the passage of undigested food. Jaundice may result from duodenal catarrh. The abdomen is protuberant and tympanitic, its enlargement being mainly due to the spleen which may extend as low as the left iliac fossa. Occasionally a bruit is heard over the tumour. Perisplenitis sometimes occurs and attacks of paroxysmal colicky pain in the left hypochondrium, due to traction on the sympathetic plexus or twisting of the peritoneal attachments. The liver usually extends an inch or two below the costal margin. The lymph glands attain the size of a pea or small bean in half the cases. Hæmorrhages are not frequent, though epistaxis and petechiæ are liable to break out in the course of the disease and gastro-intestinal bleeding in late stages. The urine may contain a trace of albumin. Pyrexia is variable, irregular, and commonly set up by complications.

The *blood* shows the changes found in chlorosis and polymorphism. The number of red cells is usually reduced to 2-3 million, or even to 800,000. The percentage of hæmoglobin falls to 30 or lower, and is more reduced than the number of red cells. The colour index is low. There are many microcytes, megalocytes, poikilocytes and polychromatophilia. Megaloblasts and normoblasts are present. Karyokinesis is common and regarded by some observers as of diagnostic importance. The number of white cells is very variable. Leucocytosis is probably due to some complication. These cells exhibit great variety in size, shape and staining properties; the variation being more marked in the polymorphs than the lymphocytes. Eosinophiles are decreased, normal or increased in number. Mast cells are often found and myelocytes in small numbers, and in even large numbers in late stages. There is generally a relative lymphocytosis, such as is common in all infantile ailments, unless there are complications. Thus the blood picture is that of chlorosis and a reversion to a more infantile type.

*Diagnosis.*—A pure case is one with no history or evidence of syphilis or rickets. Anæmia and splenic hyperplasia are rarely great in congenital syphilis and commonly found under 6 months of age. The blood picture affords comparatively little reliable assistance but a certain amount of stress can be laid on the presence of a large number of hæmatoblasts, karyokinesis, poikilocytes, polychromatophilia, leukopenia and a relative



lymphocytosis. I think leucocytosis is merely a sign of coincident disease. The changes in the red cells occur in any form of grave anæmia, and leucocytosis is also often present in such affections. The presence of a large number of myelocytes may lead to an erroneous diagnosis of true leukæmia, but this disease is very rare in infancy and there is no evidence that it is ever a sequel of the milder affection.

The *prognosis* is uncertain for cases are chronic and apt to be lost sight of. They tend to relapse, but on the whole the outlook is good and probably about 75 per cent. recover. Some patients remain stationary or even get gradually worse for 2 or 3 months, and then suddenly take a turn for the better and rapidly recover. No case is hopeless. Gastro-intestinal complications increase the gravity of the prognosis. (Edema of the feet and petechial hæmorrhages on the abdomen and trunk are bad, though not necessarily fatal indications. Death is usually due to intercurrent disease, notably bronchitis, broncho-pneumonia or miliary tuberculosis. It may result from marasmus and simple asthenia. The percentage of leucocytes is of no value in prognosis for a progressive increase may occur in fatal cases or leucopenia be present throughout. In a boy, aged 13 months, extremely ill, a large number of myelocytes were found yet, in spite of the profound blood changes, he eventually recovered. It is probable that the presence of myelocytes in any large number is an unfavourable sign. The number of hæmatoblasts is no measure of the severity. In cases which recover, the spleen may contract to its normal size. Possibly some terminate in the splenic anæmia of later life. It does not develop into true leukæmia.

**Splenic Anæmia** in older children is closely allied to anæmia splenica infantum. It is characterised by splenic enlargement, leucopenia, blood of a chlorotic type, no glandular hyperplasia, and a prolonged course with liability to hæmorrhages, especially from the gastro-intestinal tract. A case of this nature was a girl, seen in consultation with Dr. Macintosh of Hampstead. At 5 years of age she had hæmatemesis. At 9 years, when she came under Dr. Macintosh's care, she was anæmic and had an enlarged spleen. Next year she had hæmatemesis and melæna, and the spleen reached the umbilicus. The blood showed considerable reduction in red cells, marked leucopenia (about 2000) and a relative lymphocytosis of small cells (55 per cent.).

Similar cases have been recorded in more than one member of a family : 6 cases in 2 generations, two of them children 7 and 10 years old, had anæmia, leucopenia, and enlarged spleen but no hæmorrhages ; splenectomy was successful in two (Springthorpe and Stirling, 1904) ; 6 cases of enlarged spleen in 3 generations (Claud Wilson), and others. Some of these are described under the name of "family splenomegaly" and are associated with dwarfing, infantilism, clubbing of the fingers, sometimes hepatic enlargement and skin pigmentation. In family acholuric jaundice (p. 136) the spleen is generally enlarged.



Banti (1881) differentiated a special variety under the name of *progressive anæmia* or *anæmia ganglionare*. Its features are a progressive anæmia, disturbance of organic functions, irregular fever, hæmorrhages from the stomach and bowels, splenic enlargement, and a fatal termination. Both the liver and spleen show fibrosis, and there is a black or steely grey pigmentation of the skin. The course of events is first a primitive splenomegaly with no disturbance of health; a stage of splenic anæmia, secondary anæmia and leucopenia; and a final stage of hepatic cirrhosis, jaundice and ascites. The affection has been regarded by Rolleston as a special entity due to endothelial proliferation in the spleen. Most of the cases are more probably atrophic cirrhosis with splenic enlargement, syphilitic cirrhosis of the liver and spleen, or of cirrhosis occurring in the course of splenic anæmia.

*Lymphatic Anæmia* was described by Alex MacGregor (1901) as a special variety of anæmia. Of 23 cases from 3-16 years old, 18 were under 11 years. The glands, chiefly in the groins and cervical triangles, are enlarged, small and hard. The spleen is usually enlarged. The symptoms are anæmia, clean tongue, poor appetite, constipation, loss of flesh and cough, worst at night. The blood is deficient in red cells and hæmoglobin; and shews a lymphocytosis of large cells:—small lymphocytes 12-13, large lymphocytes 20-40, polymorphs 41-63, eosinophiles 2-7 per cent. Further observations are required before this can be separated as a special disease. All the cases recovered. It is probably a variety of secondary anæmia.

**Leukæmia** was formerly divided into lymphatic and splenic types. Subsequently the importance of the bone marrow was recognised in the splenic type and it was named spleno-medullary, and later on medullary or myelogenic leukæmia. More recently it has been realised that it cannot be sharply differentiated in this manner. Lymphatic cases occur in which the lesions are limited to the bone marrow; and myelogenous changes have been found in adenoid tissue in myelocytic cases. The development of lymphoid cells from adenoid tissue and of granular cells from the bone marrow is not strictly limited, and vicarious action takes place.

Clinically it is useful to recognise a *Lymphocytic* or lymphoid variety, and a *Myelocytic* or mixed cell type, although the classification is based on the blood picture only. The lymphocytic type is called *Lymphæmia* or *Lymphocythæmia*, and the myelocytic is known as *Myelæmia* or *Myelocythæmia*. Both may occur in the so-called spleno-medullary form of the disease. Myelæmia can occur without either glandular or splenic enlargement; and lymphæmia may be present without enlarged glands but with an enormous spleen. The bone marrow is always involved and the glandular system may escape entirely. The essential features of leukæmia generally are leucocytosis, a relative alteration in the percentages of normal cells or the presence of abnormal ones, and changes in the bone marrow, spleen and lymph glands.



*Acute Lymphæmia* is the variety of leukæmia seen most often in children, though it is quite a rare disease. Only about 25 cases are on record. Emerson (1907) recognised 4 different varieties:—(1) An acute infectious type, like an acute streptococcal pyæmia but without streptococci; (2) Cases like chronic leukæmia but more acute; (3) Hæmorrhagic; (4) Acute cachectic cases. Its etiology is doubtful. Possibly it is due to toxæmia or acute infection. Although streptococci have been found in some cases they were probably a terminal infection. The enlargement of the spleen is not an essential feature, and probably neither the spleen nor the glands are primarily involved. The glands are generally enlarged. The bone marrow shows lymphadenoid degeneration, a reversion to the foetal condition, and lymphæmia may be intense when the spleen and glands are unaffected. Hence the lymphæmia may be due in some cases to proliferation of the non-granular cells in the marrow, and in others to overstimulation of lymphoid tissues throughout the body. The evidence is in favour of it being a primary disease of the bone marrow.

The onset is insidious with pallor and languor. Anæmia is progressive. The hæmorrhagic tendency may be present from the first or follow profound anæmia and general glandular enlargement. Infective purpura is possibly an acute leukæmia. Some cases simulate scurvy, because of the predominance of mouth symptoms. Usually the glands, liver and spleen show some enlargement, varying during the course of the disease; and often there is slight albuminuria. The temperature is generally raised and irregular in acute stages.

Leucocytosis is commonly, but not always, present and may be extreme; occasionally there is leucopenia. The lymphocytosis is one of large cells, rarely of small cells, and is absolute or relative. With extreme leucocytosis the small lymphocytes may in rare cases predominate; otherwise the large lymphocyte is in excess. A differential count shows as many as 97 per cent. lymphocytes, few polymorphs, rarely eosinophiles. Hæmatoblasts and myelocytes have been found in small numbers in most recent cases. The number of red cells drops to 1-2 millions and the hæmoglobin index may be above or below normal.

These patients die in a few days to 6 weeks. Post-mortem examination shows general lymphocytic infiltration, hæmorrhages, a pale liver and much enlargement of the kidneys. The marrow is affected as in myelæmia.

*Chronic Lymphæmia* shows general glandular enlargement, excessive leucocytosis, and predominance of the small, rarely of the large lymphocytes. It is still more rare than the acute type. Mixed varieties may occur.

*Myelæmia* is acute or chronic. Hirsch and Cabot have recorded acute cases. About 7 cases of the chronic type have been found in children. The spleen is large and contains an excess of lymphoid tissue. The liver shows similar changes. The mesenteric glands are enlarged but the others are little or not at all affected. The marrow, especially of the long bones,



is of the "red currant jelly" appearance or, more rarely, gelatinous and puriform. It contains hæmatoblasts, myelocytes and all sorts of red cells. The thymus is generally large. The blood contains 20-60 per cent. myelocytes, many polymorphs and few lymphocytes. Mast cells are present. It resembles *café au lait*, and serous and visceral hæmorrhages are often present.

The symptoms are progressive weakness and increasing pallor; increase in the size of the spleen and liver; shortness of breath, perhaps paroxysmal dyspnœa; dyspepsia and attacks of diarrhœa; fainting attacks; bleeding from the nose, stomach, bowel, gums, lungs, kidneys, etc.; anasarca and ascites in late stages; moderate irregular pyrexia; albumin and casts in the urine, and occasionally attacks of colic due to the deposition of uric acid in the kidneys. The prognosis is worse in lymphæmia than in myelæmia. In adults myelæmia sometimes runs a very slow course. Insufficient cases have been recorded in children to enable an approximate duration of life to be fixed. Few cases of lymphæmia are likely to live longer than 3 months. Life may be more prolonged in the other type and death is often due to intercurrent disease.

**Chloroma** is a rare disease in many respects allied to acute leukæmia. It was described by Alan Burns in 1823, and first diagnosed during life by Melville Dunlop in 1901. About 50 cases are on record, over three-fifths in males. Some of these were in children 3 and 4 years of age. The affection is most common before puberty. The characteristic features are severe anæmia, prominent eyes or exophthalmos, one or more tumours with a greenish tinge in the orbits, temporal fossæ and cranial periosteum, and a marked lymphocytosis of large cells. Some cases are ushered in with facial paralysis; pain in the forehead, top of the head or mastoid region; exophthalmos, proptosis, chemosis, and perhaps optic neuritis and retinal hæmorrhage; dilatation of the veins of the eyelids and temples; œdema of the eyelids, face and forehead; and semi-fluctuating swellings in the temporal regions or hard immovable tumours here, in the mastoid region and other situations. The child has a peculiar yellowish or greenish pallor, and gradually gets weaker and dies from exhaustion in a few weeks or months. The symptoms vary with the size and distribution of the tumours. Drowsiness, semi-coma, epistaxis, petechial hæmorrhages, bleeding from the orbit, destruction of the cornea and the formation of a fungating tumour, and other cutaneous and mucous hæmorrhages have been noted. The spleen and glands are at times enlarged.

Post mortem, the growths have been found arising from the pericranium, over the outer surface of the dura mater, in the basal fossæ, in the ethmoido-facial region of the skull, beneath the periosteum on the under surface of the sternum, spine and ribs. The bone marrow, spleen and glands are infiltrated. Metastases are present in numerous places, e.g., liver, kidneys and ovaries. The tumours are greenish in colour, from a colour like that of turtle fat up to bright green. Some of them are not



coloured. The cause of the green tint is not known. The disease appears to start in the red marrow of a cranial bone and less often in that of a vertebra, rib or the sternum.

Clinically these cases are very much like those of sarcomata of the skull, orbits and adrenals (p. 211). Microscopically the structure is that of an infiltrating round-celled sarcoma. The blood picture is that of lymphæmia. Possibly the large mononuclear cells found are really modified myeloblasts; and the disease is a variety of myelogenous leukæmia. Treadgold (1908) suggests there are three stages or three different affections, viz., Sarcoma of bone; Chloroma, a tumour of myeloblasts, arising primarily in the marrow, remaining focal for a time and producing metastases; and Acute Lymphæmia, a tumour also of bone marrow, consisting of amphoblasts, leucoblasts or mixed cells.

*Treatment of Anæmia and Allied Affections.*—Diet and hygiene are necessary and the primary cause, if discoverable, must be treated. In severe cases rest in bed is essential. Iron and arsenic are the best drugs. Iron can be given in organic forms in yolk of egg, bone marrow, raw meat and raw meat juice, potatoes, spinach and other foods (p. 34); or in inorganic preparations (p. 81). One gm. of inorganic iron is present in reduced iron 1, saccharated carbonate of iron 10, tr. ferri. chlor. 28, iron somatose 50, carniferrin 3·3, triferrin 4·5, ferratin 15·4, hæmatogen 1420, hæmoglobin 233, hæmogallol 359 gms., and in 5 Blaud's pills. Arsenic is useful in pernicious anæmia, when given in full doses, and is often beneficial in leukæmia, in which it may reduce the number of white cells without increasing the erythrocytes.

Sea air and high altitudes are not as good as country air. Avoid chills, loss of body heat, exposure to the direct rays of the sun and sea bathing. Moderate massage can be used to replace exercise.

The removal of the spleen in anæmia splenica infantum is unnecessary and useless; in leukæmia it is soon fatal. In the family form of splenomegaly and Banti's disease it may be followed by recovery. Harris and Hertzog (1901) collected 19 cases of which 4 died. After the operation there is some temporary fever and leucocytosis and the blood condition improves. A gradual compensation has taken place during the course of the disease, so that the operation is not very serious. The treatment of leukæmic cases by exposure of the enlarged spleen to the action of X-rays has occasionally proved beneficial.

**The Lymph Glands** consist of adenoid tissue and lymph sinuses. According to Warthin (1902) hæmolymph glands, containing blood sinuses instead of lymph sinuses, are found in the prevertebral retro-peritoneal tissue, usually near the great vessels; less often in the mediastinum; and frequently near the parathyroids. Possibly they are compensatory to the bone marrow and spleen in function. Of these the *splenolymph* glands are the more common, and may be mistaken for secondary spleens. Their



lymphadenoid tissue contains small and large lymphocytes, transitionals and polymorphs; rarely mononuclear eosinophiles and mast cells. The sinuses are lined by endothelium and contain red and white cells. They are chiefly hæmolytic and partially leucocyte-forming organs. The *marrow lymph* glands contain blood sinuses filled with a coarse reticulum through which the red cells filter. Masses of lymphoid tissue are arranged between the sinuses but there are no true follicles. Many scattered fat cells are present. The lymphadenoid tissue contains mononuclear eosinophiles, polymorphs, occasionally myelocytes of bone marrow type, erythrocytes and deposits of hyaline material. Their function is the formation of leucocytes and they take little part in hæmolysis.

*Glandular Enlargement* is caused by proliferation of the lymphadenoid and connective tissues. It varies in consistency, and may be very hard or may undergo softening, caseation and suppuration. It is induced by the irritation of toxins or micro-organisms which reach the gland through the lymphatics or blood stream, commonly the former. Adenitis causes leucocytosis of polymorphs and sometimes eosinophilia. Always seek for some local source of irritation, bearing in mind the distribution of the lymphatics running to the affected gland. In many instances the enlargement is protective and a measure of the activity of the lymphocytes in the gland in destroying the microbes or toxins which reach its meshes. The glands in this way act as a barrier to the spread of the infection into the system. Sometimes the process is very acute, the poison strong, and the infection one by pyogenic organisms, so that the gland undergoes suppuration.

According to the situation of the glands there are different causes of enlargement. Some causes produce general lymphatic hyperplasia, e.g., Hodgkin's disease and, to a less extent, specific fevers, and the continued toxæmia of prolonged tuberculous or septic affections. More usually the enlargement is limited to a group of glands, especially those in the cervical regions, not a surprising fact when the numerous sources of infection about the scalp, ears, face, nose conjunctivæ, mouth, teeth and throat are taken into account. Occasionally no definite cause can be found, as in those cases which follow exposure to cold and in the so-called glandular fever. Possibly some of these are due to the rheumatic poison. Cervical adenitis has been noted in cases of erythema nodosum, and this has sometimes been thought a rheumatic manifestation, though it is much more probably toxæmic.

Cervical adenitis from throat and mouth affections differs in its location. The submaxillary lymph nodes are affected as the result of carious teeth, if the gums are inflamed; and later on the gland below and behind the angle of the jaw is attacked. This is distinguished from the submaxillary lymph node by being more movable posteriorly. It is the one which becomes enlarged in affections of the faucial tonsil; and subsequently the lower deep cervical glands may become enlarged. In diseases of the pharyngeal tonsil the glands just beneath the posterior border of the



sterno-mastoid are first attacked, then those under the muscle, and lastly those in front of it, causing a diffuse swelling of the upper part of the muscle.

Tuberculosis is one of the most common causes of adenitis, and is particularly liable to follow enlargement from other causes. The vitality of the gland is lowered and the tubercle bacillus finds in it a suitable nidus for its growth and development. Tuberculosis of glands is therefore due to primary infection of a patient unduly susceptible to tuberculous disease; to secondary infection of a gland of lowered vitality; or to a mixed infection by pyogenic organisms in addition. In the last case there is a great tendency to suppuration. The effect of tuberculous infection is first hyperæmia and swelling; secondly, proliferation of the adenoid reticulum; and finally destruction or conversion of lymphoid cells and caseation. Sometimes there is merely hyperplasia. The tubercle bacilli may be found in involution forms, known as "Schrön's capsules." Several glands are generally affected. In the early stages they are hard and freely movable on themselves and the surrounding tissues. Later they become adherent to each other and to the skin, and often break down. Softening is due to caseation or pyogenic infection. The skin becomes involved, inflamed, thin and perforated, and the pus and caseous matter are discharged. Occasionally the tuberculosis starts as an acute process. Much more often it begins quietly, remains more or less stationary for some time, and then undergoes rapid exacerbation and breaks down. For this reason it is imperative to keep such cases under close observation, as the time for operation is before the skin and subcutaneous tissues have become involved. Tuberculous supracondylar glands are due to strumous dactylitis and the inguinal glands may become infected by circumcision.

*Generalised Tuberculous Adenitis.*—A few cases have been described in which many glands are acutely involved, possibly as the result of a general infection of the glands with the tubercle bacillus. They run a short course and exhibit anæmia, irregular fever with exacerbations, and polymorphonuclear leucocytosis. The spleen is little or not at all enlarged.

**Tuberculous Thoracic Glands.**—The bronchial, anterior and posterior mediastinal glands may be enlarged separately or simultaneously, almost always from tuberculous infection. Very considerable enlargement is needed to produce symptoms; and hyperplasia, caseation, fibrosis and calcification can occur without any evidence thereof. Enlargement of the anterior mediastinal glands may be so great as to cause dulness on percussion over the manubrium. According to Durham these glands are commonly the first affected in peritonitis, for they are the most important drain of the peritoneum. If so, it is curious that they are not more constantly involved in tuberculous peritonitis. Enlargement of the posterior mediastinal glands may produce bronchophony, bronchial breathing and then dulness high up in the interscapular region.



The tracheo-bronchial glands are those situated about the root of the lungs, between the divisions of the bronchi. They are usually all affected; more especially the "pre-tracheal gland," the one at the bifurcation of the trachea. If the disease is unilateral, it is 4 or 5 times more common on the right side than on the left, possibly because the right bronchus is greater than the left. The tuberculous infection is conveyed to the bronchial glands by the inspired air; by the pharyngeal lymphatics from the pharynx, tonsils and adenoids, via the cervical and mediastinal glands, thoracic duct and right side of the heart; from the intestines, mesenteric and retro-peritoneal glands; and occasionally from the œsophagus, lungs, carious vertebræ or by the blood stream. In quite three-fourths of all cases of miliary tuberculosis and tuberculous meningitis in early life the oldest tuberculous lesion is in the tracheo-bronchial glands, generally in the pre-tracheal gland, and it is exceptional to find evidence of a cervical adenitis. This is an important point for it is an argument against the entrance of the tubercle bacillus into the system through the mucous membrane of the mouth and throat.

The *symptoms* depend on the degree of engorgement and the situation of the affected glands, and may be equivocal or even temporary. A rise of temperature in the evening is not infrequent and the child may waste, be easily tired, complain of pain under the manubrium and have a troublesome cough. The cough is due to pressure or irritation of the recurrent laryngeal nerve. It is spasmodic, paroxysmal, suggestive of but more constant than pertussis, and occasionally ends in vomiting. Pressure on the trachea or bronchus may cause dyspnœa on slight exertion and stridor. If on a bronchus, it may render the breath sounds weaker over the corresponding lung. The irritation from pressure may set up bronchitis, broncho-pneumonia and bronchiectasis. Occasionally the gland ulcerates through into the trachea or bronchus and causes sudden death from asphyxia, or severe and fatal hæmorrhage, a small circular ulcer being found after death. Sometimes the caseous material is coughed up and the child gets well. Ulceration into the air passages gives rise to offensive breath. The pericardium may be invaded and ulceration into the aorta has occurred.

Pressure on the œsophagus is indicated by dysphagia. The pre-tracheal gland is apt to perforate the œsophagus. A girl died from hæmorrhage, blood and air being brought up, after an illness of 9 months with wasting, diarrhœa and vomiting, and offensive breath. The stomach was full of blood and a small circular ulcer of the œsophagus was found. It had smooth, sloping edges, and was secondary to an abscess of the pre-tracheal gland which opened also into the lower lobe of the right lung. Similar cases are on record and others in which the gland had only ulcerated into the œsophagus. Rupture into the lung is generally followed by extensive caseous pneumonia and general dissemination. In children it is not very uncommon to find tuberculous infection spreading backward along the



lymphatics running into the gland, causing a definite triangular patch of consolidation in the lower lobe. Occasionally the gland ruptures into the pleura and sets up empyema. In a child, aged 4 years, there were signs of enlargement of the anterior mediastinal glands, dulness and bronchial breathing over the manubrium. Gradually the dulness extended until the note over the whole right upper lobe was impaired. Exploration high up in the axilla revealed the presence of pus. The abscess was opened and a probe could be passed upward and inward, between the upper and middle lobes, in the direction of the manubrium for 3 ins. The child recovered. Tuberculosis of the lungs, especially of the lower lobes, in children is commonly a sequel of tuberculous adenitis. The caseous glands may become inflamed as the result of non-tuberculous disease and then produce general dissemination.

*Physical signs* are sometimes well marked. They are generally due to pressure on veins and to the size of the enlargement. Pressure on the vena cava superior causes dilatation of the veins in the neck and on the cheeks, temples and front of the chest; puffiness of the face and eyelids; œdema and cyanosis; headache and attacks of giddiness. These signs may be unilateral, if the innominate vein alone is compressed. A continuous venous hum, heard over the manubrium when the head is thrown back, has been ascribed by Eustace Smith to pressure on the vena cava superior or the left innominate vein. It is not always present and may occur though no enlarged glands are found after death. Enlarged glands under the manubrium cause dulness which extends laterally, especially to the right, and bronchial or tracheal breathing. Enlargement of the posterior mediastinal glands causes dulness on either side of the spine, from the second to the fifth dorsal vertebra, prolonged expiration, bronchial or amphoric breathing, and bronchophony, notably on the right side. Normally the bronchial tone stops at the seventh cervical spine. If the glands are enlarged, it may extend lower, even down to the fourth dorsal spine. X-ray examination may show an increase in size and density of the shadow at the root of the lung. Lymphocytosis is sometimes present. The subclavicular and episternal lymph glands may be enlarged. Undue hairiness of the chest has been noted. Anæmia, anorexia, night sweats, epistaxis, fever and hæmoptysis may occur.

**Tuberculous Mesenteric Glands.**—The name of “*tabes mesenterica*” should be limited to the rare cases in which the mesenteric glands are tuberculous, apart from peritonitis. It is a rare disease in that it is not often diagnosed unless the peritoneum is also involved. A movable tumour in a child’s abdomen is much more likely to be a fæcal mass than an enlarged gland. In the early stages, before general infection or peritonitis occurs, the condition is not found for no attention is attracted to the abdomen. These lumps are found in children admitted to hospital for general ill-health, abdominal distension, slight fever, anorexia and constipation.



They are due to primary infection or tuberculous enteritis; and probably the cause, but not the result of tuberculous peritonitis. The glands may calcify, become caseous, set up caseous peritonitis, and rarely suppurate. Whitworth (1908) reported a case of ulceration into the superior mesenteric artery. Adhesions, intestinal obstruction and local peritonitis may ensue. The most common situation is to the right of and a little above the level of the umbilicus, or below and to the right, and less often below and to the left of the navel.

Symptoms may be absent. Occasional signs are anæmia, pain and vomiting; colicky pains and loose stools; wasting and jaundice, due to pressure or of toxic origin; and enlargement of the liver. They must be diagnosed from subacute intussusception, fæcal masses, hydatids, new growths and caseous peritonitis. Caseous masses are included among adherent gut and are little, if at all, movable.

*Treatment of Adenitis.*—In the treatment of all visible or palpable glands it is essential to remove or treat any local exciting cause of irritation such as carious teeth, alveolar abscess, adenoids, enlarged tonsils, throat affections, ear troubles and skin lesions. If there is evidence of tuberculosis rely on general treatment by sea air, hygiene, etc., and give cod-liver oil, maltine, syr. fer. iodidi, ichthyol and ferri-ichthyol. Friction with ointments is not advisable for it may cause irritation and induce suppuration. Painting with iodine is useless. An ointment of ung. hydragyri, ung. belladonnæ and olive oil, p.a., may be smeared on. Operate before the glands become adherent or break down, provided that they are undoubtedly tuberculous. Little scarring is left. The removal of tuberculous cervical glands is not an operation to be undertaken lightly for many more glands will be found affected than can be felt, and they must all be thoroughly and carefully removed. The removal of those chiefly affected is rapidly followed by enlargement of others left behind, and further operation is needed. To incise and scrape a tuberculous gland is bad treatment. A sinus is almost certain to be left which heals with difficulty, and the skin and surrounding tissues may become infected. It is doubtful whether it is ever advisable to attempt the removal of tuberculous mesenteric glands, though it has been carried out successfully. Most cases get well under medical treatment.

For inflammatory enlargements use hot or cold applications, belladonna and glycerine, or mercury and belladonna ointments. Local treatment must be applied gently.

**Glandular Fever.**—*Syn. : Drüsenfieber (Pfeiffer), Febrile Polyadenitis, Fièvre Ganglionaire.*—Pfeiffer (1889) suggested the possible existence of glandular fever as a distinct entity, and many cases of this type have been recorded since. It is an infectious disease, running through a household and especially affecting the younger members. It occurs at any age, commonly 4-12 years, and is infrequent after puberty. Both sexes are equally liable. The incubation period is stated by Byers to be 5-7 days.



The onset is sudden, often with headache, abdominal pain and vomiting, perhaps with convulsions or rigors. The temperature rises to 101-104° F. In 24-48 hours the glands at the angles of the jaws and behind the sternomastoid muscles, forming a regular chain, are enlarged. They are tender, painful, hard, movable, of the size of large beans, and cause discomfort and stiffness of the neck, and perhaps dysphagia. They may even reach the size of a pigeon's egg. The skin is rarely reddened. The adenitis may not commence until the third or fourth day, and may be unilateral. It continues for a few days to a week, gradually subsides in 7-10 days, and may last 4 weeks. The fever is from 1-2° F. lower in the morning than the evening, and is of short duration. With it are associated from the onset headache, foul tongue, anorexia, nausea, sometimes vomiting and constipation. In mild cases there may be diarrhœa. Abdominal pain is a prominent symptom but may be absent. The face is flushed in the febrile stage and afterwards unduly pale. The liver and spleen are enlarged in about three-quarters of the cases in some outbreaks. The mesenteric, axillary and occasionally the inguinal glands are enlarged. Undue redness of the fauces and pharynx has been found sometimes, even sorethroat at the onset, but no definite pharyngitis or tonsillitis.

The chief complications are epistaxis and hæmorrhagic nephritis; simple albuminuria; the passage of urine containing hyaline, epithelial and granular casts; or simple hæmaturia without any evidence of nephritis. In two boys hæmaturia was the main sign; in another, who had just escaped operation for the removal of glands because albumin was found in the urine, a relapse a few days later was accompanied by hæmaturia. The glands do not suppurate.

The disease is mild if the temperature does not rise above 102° F., and severe if it reaches 104° F. Death is a rare event and due to some complication. The fever lasts from 3-7 days and may end by crisis and sweating; or the illness may last from a few days to a fortnight with irregular pyrexia. The adenitis subsides slowly. The renal complications in cases under my care have invariably cleared up entirely. Convalescence is prolonged and characterised by anæmia, debility and depression.

At the onset the illness simulates influenza or a specific fever, notably aberrant typhoid, or some throat affection. Mumps can be excluded, for the parotid glands are unaffected and there is no pain on eating. Local sources of adenitis must be looked for and tuberculosis, Hodgkin's disease and the early stage of acute leukæmia borne in mind. It is doubtful whether we are justified in regarding this as a specific disease though it is very convenient to do so. The course, symptoms, complications and delayed convalescence are very suggestive of a microbial infection of the throat or pharynx, perhaps post-nasal. Pneumococci and streptococci have been found. The treatment consists of cold compresses, belladonna fomentations, or belladonna and glycerine locally; mild purgation by



calomel ; phenacetin, salicin, quinine or a mild febrifuge ; bed and light diet ; tonics, iron and sea air during convalescence.

**Lymphosarcoma.**—This is a malignant disease of the glands, sometimes seen in children from 2-15 years of age. It begins in mediastinal glands, generally the posterior ones, and spreads to cervical glands, in which it is first evident or possibly at times primary. In the earliest stages, when cervical enlargement is alone present, the lump is sometimes called a *Lymphoma*. This stage lasts from one to several months without malaise, pain or local discomfort. Many glands in series are affected, on one or both sides ; and are firm, of uniform consistence, discrete, movable, and neither painful nor tender. The skin is uninvolved. Subsequently the child develops anæmia, anorexia, debility, shortness of breath, cough, faint or giddy attacks, and choking fits, from pressure on various intra-thoracic structures. The symptoms and physical signs are those of enlarged thoracic glands, as above described. The glands tend to become fused, though there is no sign of inflammation. In late stages the spleen may be enlarged, the axillary glands rarely involved, and ascites and œdema develop. The blood picture is that of mild anæmia, with leucopenia or a relative lymphocytosis. Microscopically the growths consist of masses of small round cells, like lymphocytes, with large nuclei and a fine stroma, very little vascular. In the case of a boy, 3 years old, the growth was apparently a round-celled sarcoma of a posterior mediastinal gland, no cervical or other glands being affected. It compressed without invading the right lung, which was practically airless. The tumour had broken down in the peripheral portion, forming a large irregular cavity filled with blood and clot. It was adherent to the diaphragm and chest wall, except over the upper lateral and anterior portions. In another 3-year old boy, who died during an attack of dyspnœa and cyanosis from tracheal pressure, the growth was retro-pleural and had apparently started in the left œsophageal or posterior aortic glands in the upper part of the mediastinum. The duration of these cases is very variable. From the time that symptoms due to the intra-thoracic growth develop to the fatal termination, the illness lasts from a few weeks to several months.

**Lymphadenoma or Hodgkin's Disease** differs from lymphosarcoma in that it affects all the lymphoid tissues and the glands remain distinct. It is rare in infancy and childhood. Many of the cases described under this name are probably really instances of lymphosarcoma, multiple tuberculous adenitis and possibly syphilis. Out of 43 cases 10 were under 10 years of age (Mitchell Clarke, 1901). Hutchison (1904) states that it is rare under 5 years of age. Greig (1907) says that it occurs after 30 years of age.

The signs are anæmia and progressive hyperplasia of the lymph nodes. The liver and spleen are usually enlarged and contain lymphoid masses resembling secondary growths, and similar masses may be found in other organs. Limited to one group of glands it may be spoken of as a *Lymphoma*. Acute cases show moderate fever and a hæmorrhagic tendency, suggestive



of an infectious origin. When generalised it resembles chronic lymphatic leukæmia clinically, except in its blood picture. At first the blood is normal. Later, it shows the usual characteristics of secondary anæmia. Polynuclear leucocytosis may be present from secondary infection, and a few myelocytes and hæmatoblasts in advanced cases. The glands are firm, movable, non-adherent, and do not break down unless there is secondary infection. They are found in the neck, axillæ, groins, mediastinum and mesentery.

Greenfield (1877) recognised hard and soft types, due to chronic inflammation of the fibrous stroma of the glands. Andrewes (1902) stated that the distinction between the cortex and medulla is abolished, the stroma hyperplastic, lymphocytes decreased, eosinophiles generally increased, and the endothelial cells large and multinucleated (lymphadenoma cells). D. M. Reed (1902) found proliferation of endothelial cells and dilatation of blood vessels, and that the spaces were filled with these cells and masses of lymphoid cells in early stages; the fibrosis being a later development.

Lymphadenoma is sometimes differentiated from Hodgkin's disease. It is probable that some of the cases are identical therewith and others are lymphosarcomata. Rolleston and Latham found the stomach of a boy, 18 months old, covered with numerous smooth white polypoid growths of lymphoid tissue, enlarged anterior mediastinal glands, large spleen, and a blood picture of myelogenous leukæmia. It was supposed to be secondary to ear disease. In a boy, 14 months old, with otitis media, masses of enlarged non-adherent glands were present on both sides of the neck and the spleen reached the crest of the ilium. The blood count yielded red cells 7,470,000, whites 60,000, small lymphocytes 14·4, large lymphocytes 31·5, polymorphs 53·9 per cent. and a few eosinophiles. The autopsy showed many cervical glands breaking down from secondary infection, enlarged mesenteric glands, a mass behind the sternum and at the root of the lung, fatty liver, fibrosis of the spleen, no leucocytic infiltration, and giant cells in the liver, spleen and lungs. The glands in lymphadenoma seem specially prone to secondary tuberculous infection. Cases of the above type indicate that the differentiation into distinct groups is difficult and that there are probably intermediate varieties or mixed infections. It is not uncommon for Hodgkin's disease to start as a secondary process, after a primary disease of the ear with glandular infection. Tuberculosis may be a further complication. Moreover tuberculosis is not a rare sequence of anæmia splenica infantum, and secondary tuberculous adenitis or a lymphadenomatous condition in the course of this disease would give rise to grave difficulty in diagnosis.

The prognosis is bad except when the mischief is limited to one group of glands. Leucocytosis, due to secondary infection, may suggest the onset of acute leukæmia, an occasional termination. The treatment is on the same lines as that of other types of anæmia.

**The Thymus.**—The thymus is a ductless gland found in all true



vertebrates and demonstrated by Schaffer in marsipobranchiate fishes. It is reddish-grey in colour, situated in the anterior mediastinum just above the pericardium, and extends upward to about 2 cm. above the sternum and 2 cm. below the thyroid, lying between the innominate and left carotid arteries. It is thickest behind the manubrium and there covers part of the vena cava superior, the innominate veins and the arch of the aorta, and extends backward to the trachea. Lower down it is partially covered by the anterior edges of the lungs. In transverse section of an infant it may occupy as large an area as that of the left lung. It may cause marked dulness on percussion, continuous with the cardiac dulness, and extending more to the left than the right. It receives blood from the inferior thyroid arteries and its nerve supply from the sympathetic.

It is somewhat flat, composed of two lobes, usually asymmetrical and united by an isthmus, and covered by a fibrous capsule from which septa pass inward dividing it into lobules and follicles. Each follicle is more or less polyhedral, and consists of a dense cortical and a looser medullary portion, dependent on the number of lymphoid cells. In the medullary parts are seen the concentric corpuscles of Hassall, protoplasmic masses with a nucleated granular centre surrounded by flattened cells. They are the remains of original epithelial outgrowths. Eosinophiles are fairly numerous. The size and weight of the gland are very variable in different infants and at different ages. It varies in the young of the same litter, is rarely absent except in acephalic twins, and is often large in monsters. It is normally largest shortly after birth, remains about the same size for 2-5 years, and then slowly atrophies. The avian thymus does not undergo regressive changes. As it atrophies it is converted into fibrous tissue, and may be infiltrated with fat after puberty. The weight is no reliable guide to the functional capacity. No constant ratio to body weight can be made out.

The *weight* in grammes at birth is said to be 3-5 by several observers, 8-12 (Haughsted), 13-20 by others. On the whole it is probable that the lower figures are the more reliable, and that the other ones are due to including exceptional weights. Similarly I think we may accept 5-7 gms. as an average weight during the first 5 years of life and regard glands of greater weight as to some extent abnormal.

Its *functions* are somewhat uncertain. The gland is hypoblastic in origin, being developed from the epithelium of the third pouch of the gut-tract, and acquires its infantile type in the third month of foetal life. Kölliker regards it as the parent source of leucocytes. For a relatively long time the blood only contains nucleated red cells and at the period when leucocytes first appear there is no spleen, rectal gland or cæcum, and the thymus is represented by small pieces of modified epithelium. Beard suggests that the first leucocytes are formed by modification of these epithelial cells and that the thymus is the parent of *all* leucocytes. Bryce, however, states (1904) that he has found leucocytes in the blood of *Lepidosiren* before any trace of a thymus could be detected. Blood



pressure experiments with extracts of the gland seem to show that it has no direct vasomotor influence. Extirpation causes muscular weakness and progressive debility in frogs (Abelous and Billard, 1896); changes in the blood, disturbance of growth of bone, nervous symptoms and profuse sweating, a condition like rickets (Friedleben); no ill effects in dogs, except temporary weakness and erythropenia in young ones (Tarulli and Lo Monaco, 1858). In some way the gland exercises an influence on nutrition and hæmopoiesis.

*Atrophy* is noted in various conditions. The gland may be absent in the mentally defective. It is atrophied in marasmus from any cause and the weight is a fair measure of the nutrition in infancy. In moderate atrophy the cortex and medulla are not easily distinguished. In severe wasting there is much fibrosis of the capsule, trabeculæ and about the blood vessels. The endothelial cells undergo proliferation and the lymphoid cells are decreased. Small giant cells, somewhat like those seen in the glands in lymphadenoma, have been found by Dudgeon. Hassall's corpuscles stain bright yellow or orange with Van Gieson's fluid, perhaps from hyaline degeneration. In many illnesses the gland is atrophied and shows fibrosis and hyaline degeneration, often infiltration with adipose tissue. Atrophy of the thymus is part of, but not the cause of, the malnutrition.

*Hypertrophy* is present in many cases of sudden and unexpected death in infants, thymic asthma, the status thymicus or lymphaticus, in most acute conditions in which there is leucocytosis, in leukæmia and lymphadenoma, in many cases of acromegaly and gigantism, and sometimes in exophthalmic goitre, chlorosis, epilepsy and myasthenia gravis; and after castration in rabbits (Calzolari).

*Other affections* are rare. Miliary tubercles and, less often, tuberculous nodules are found. Primary inflammation has been recorded and pyæmic abscesses. Multiple minute hæmorrhages are present in asphyxia neonatorum, and scattered hæmorrhages in most lung diseases. Minute collections of a thick, yellowish-white, semi-purulent material can sometimes be squeezed out on section. They are not abscesses, but are due to softening of normal lymphoid tissue and are not pathological. Gummata are very uncommon.

**Status Thymicus vel Lymphaticus, or Lymphatism.**—As long ago as 1614 Felix Plater found the thymus enlarged in cases of sudden death from dyspnœa in 3 children of one family. That it may be a family peculiarity is shown by its occurrence in more than one child, e.g., in 3 nurslings (Weber), in 3 children (Barrock), and in 5 out of 9 children (Hedinger, 1905), an autopsy on the fifth, aged 5 years, showing the status lymphaticus. Perrin reported that 9 out of 11 children in one family died suddenly, but the cause was not ascertained. In 1889 and 1890 Paltauf collected a large number of such cases in adults and named the affection "the lymphaticochlorotic constitution." He regarded it as a morbid entity, a condition of



hyperplasia of the thymus and lymphatic apparatus generally, including the spleen. With it is associated an abnormal predisposition to sudden or unexpected death in infants and adults. A large thymus may give rise, by compression of the trachea or in other ways, to dyspnœa, stridor, asphyxia and even sudden death. This was named by Millar "*Thymic Asthma*," a title which might be reserved for those cases in which there is definite evidence of compression or, in addition, those in which attacks of dyspnœa are the chief symptom.

Clinically there are three types of these cases in which sudden death occurs. In one the child is apparently healthy and no other cause is discoverable. In the second it is associated with the presence of other disease, adequate or inadequate to produce the fatal issue. In the third a definite cause of death is present but one which is insufficient to produce it in a normal child. Such cases are those due to intense excitement or fear, a sudden cold plunge, the introduction of a tongue spatula, the injection of serum, and the action of anæsthetics. Possibly some still-births and deaths from overlaying depend on this peculiarity of the constitution. The mode of death varies. It takes the form of sudden syncope or collapse, cyanosis followed by pallor, or mild epileptiform convulsions. In thymic asthma dyspnœa, cyanosis and distress may precede a sudden termination by several hours. Other infants have laboured breathing and attacks of grunting and lividity; symptoms of croup; or attacks of suffocation with cyanosis and recession (Siegel). A common termination is for the child to sit up in its cot, turn pale or livid, and fall back dead. One child was playing quietly when he suddenly ran to his nurse and died. Perhaps while suckling or drinking out of a cup the head is thrown back and death occurs without a cry, convulsion or movement of any sort.

McCardie (1908) collected 30 fatal cases after anæsthetics: chloroform 17, ether 6, C. and E. 5, nitrous oxide 2 (doubtful cases); 1 after injection of morphine and 2 after local anæsthesia. Other cases have been reported since. The average age of 35 cases was 16 years; 7 under 10 years, and 14 from 10-20 years of age. The sexes were about equal. Death in anæsthetic cases is always sudden; with facial pallor, dilated pupils and cardiac failure, and occasionally preceded by, or simultaneous with, respiratory failure and cyanosis.

The *signs of lymphatism* are most indefinite. Many patients seem to enjoy perfect health but they are very subject to infectious disease. Usually the complexion is pale or pasty, the skin thin and pallid, and there is excess of subcutaneous fat. In addition to the enlarged thymus there may be found evidence of rickets, enlarged cervical and axillary glands, hyperplasia of the circumvallate papillæ, the tongue, the tonsils and the adenoid tissue of the pharynx, enlargement of the spleen and perhaps lymphocytosis. The heart sounds are weak, the heart often dilated, pulse



soft, and blood pressure low. The pupils are large, and there is a tendency to œdema.

*Morbid Anatomy.*—The anatomical changes are those of hyperplasia of lymphoid tissues throughout the body. In two infants, 9 weeks and 9 months old, under my care, who died suddenly from no apparent cause, the solitary follicles and Peyer's patches were so much enlarged as to suggest the early stage of typhoid fever; and the mesenteric glands were also swollen. The mesenteric and retro-peritoneal glands are especially large. Sometimes proliferation of the endothelial cells along the trabeculæ of the thymus has been found; and proliferation, with slight degenerative changes in the proliferated cells, in the spleen, lymph glands, tonsils and intestinal lymph masses.

*Pathogeny.*—Death is ascribed to mechanical pressure, cardiac paralysis, or toxæmia. Tamassia states that it requires a weight of 180 gms. to compress the trachea, and Scheele puts the weight at 750-1000 gms. Certainly there is a very narrow space, 2-3 cm. in nurslings, between the manubrium and the spinal column, and this can be further decreased by throwing the head back. Cases are on record in which the symptoms have been cured by removal of part of the gland, and the trachea has been found flattened. Evidence of compression is often absent after death, perhaps because the trachea is elastic and resumes its normal shape when the sternum is removed. It is clear that there is a small group of cases in which an enlarged thymus does actually compress the trachea and that it gives rise to attacks of dyspnœa and cyanosis, and inspiratory and expiratory stridor, mainly expiratory. The attacks are sudden, violent, and not relieved by intubation or tracheotomy. There is no evidence of compression of the heart, blood vessels or nerves.

Cardiac paralysis is undoubtedly the cause in some cases, but the predisposing factor is probably a lympho-toxæmia, not necessarily constant in action.

The *diagnosis* of thymic asthma is difficult for it varies in type. It may be fatal in the first attack or series of attacks. Sometimes there is regular sighing respiration; attacks of subacute asphyxia, ascribed to glottic spasm; or cyanosis and crowing, suggestive of laryngospasm, followed by unconsciousness, loss of reflexes, rigidity, cyanosis, and gradual cessation of the heart and breathing. Or it may end in an inspiratory crow. In other instances the breathing is rapid, cyanosis and dilatation of the veins in the neck develop, and the child dies in a few minutes from suffocation. Gradually increasing inspiratory dyspnœa is due to pressure on the trachea.

The diagnosis of enlargement of the thymus is based on the dulness on light percussion over the manubrium. It is roughly triangular in shape, with the base upward and a somewhat cone-shaped apex at a level with the second ribs. It extends a little beyond the manubrium, more to the left than the right. The thyroid gland is enlarged in about half the cases.



Sudden death may occur from numerous other causes, e.g., coryza in the newborn and pertussis, causing "swallowing of the tongue," it being drawn upward and backward and preventing the entrance of air; asphyxia from overlaying; glottic spasm, etc. (p. 102).

No treatment is available, except partial or entire removal of the gland if there is distinct evidence of tracheal pressure.



## CHAPTER XLIV.

### DISORDERS OF THE BLOOD AND LYMPH.

*Urticaria—Erythema Nodosum—Purpura—Hæmophilia.*

**Urticaria.**—*Syn. : Nettle Rash—Urticaria papulosa—Lichen urticatus—Strophulus—Prurigo.*—Many varieties are included under the title of urticaria, differing in pathology, so it is important to understand what is meant by the term. In all these affections there is a characteristic eruption of lumps, sometimes called “heat bumps,” “water blisters,” “hives,” etc., closely resembling those due to nettle stings or aggregated into wheals of similar character. At first the bumps are white and raised, involving the skin and subjacent connective tissue. In the young they quickly subside and leave small, itching, hard papules. The papules are most common on the lower limbs, forearms, wrists, hands and palms, feet and soles, buttocks and trunk, in order of frequency; especially on the extensor surfaces. Sometimes they are vesicular at the apices (*Urticaria vesiculosa*), and become pustular (*U. varicelliformis*), or they may be hæmorrhagic (*U. hæmorrhagica*), or form large bullæ (*U. bullosa*). Itching is severe and induces scratching of the parts within reach, the papules being then covered with blood crusts or scabs. If the rash becomes infected, it assumes a pustular or ecthymatous appearance.

*Giant Urticaria* is an angioneurotic œdema (p. 458). There is a rare form, epidemic in character, but the rash does not itch. *Factitious Urticaria* and *Dermatographism*, writing on the skin, can be produced in neuropathic and hysterical subjects in consequence of vasomotor instability. *Mechanical Urticaria* is the result of external irritation by plants, such as nettles and the primula obconica, insects and hairy caterpillars, notably the “woolly bear.” This variety is of particular interest in that it shows the eruption can be caused by poisonous substances which set up a vasomotor disturbance of the cutaneous vessels.

*Pathology.*—Some toxin, possibly a by-product of protein or carbohydrate digestion, is absorbed from the alimentary tract. It irritates the nerve endings and causes a rapid effusion of serum. There is a vasomotor disturbance of cutaneous vessels. The injection of diphtheria antitoxin frequently produces the rash. In acute cases the coagulability of the blood is often diminished. The itching is due to irritation of the nerve endings by toxin or by the pressure of swollen papillæ. A curious idiosyncrasy in respect of quite common articles of food is not infrequent. Thus:



an attack of acute urticaria may follow the ingestion of shell-fish, eggs, cheese, strawberries, bananas, etc. Hingston Fox (1908) suggested that the symptoms of egg-poisoning arise because eggs, like shell-fish, contain a powerful decalcifying agent, chiefly in the outer membrane. This passes from the stomach to the blood and precipitates calcium salts, thus causing diminished coagulability and transudation. Interesting cases of egg-poisoning are on record as early as the fifth month of age, the rash appearing in 6-8 minutes (Bendix). Egg produced acute urticaria, alarming collapse, swollen head and loss of radial pulse in an infant 13 months old; a second attack 2 weeks later; and a third, after custard, with severe vomiting and diarrhoea, 1 month later. Egg caused erythema exudativum bullosa in a child from the first to fifth year of life (Albu). So far I am unaware of the presence of a decalcifying agent in bananas which in my experience are sometimes the cause of acute and chronic attacks. A few cases are due to intestinal parasites.

*Acute Urticaria.*—The typical attack is seen after the ingestion of various articles of diet and shell-fish, notably mussels, the poison being probably located in the liver. Mussel poisoning gives rise to three different types of poisoning:—(1) Vomiting and diarrhoea shortly after ingestion, or after a varying incubation period with fever; (2) An acute peripheral paralysis resembling the effects of curare. It is ushered in with nausea, vomiting, constipation and dyspnoea. The face may be swollen and livid, and psychical excitement marked. The onset is variable, usually rapid and even within 30 minutes, beginning with “pins and needles” in the extremities. It varies in severity and duration, and may be quite transitory. All the muscles may be involved and death result from respiratory paralysis without loss of consciousness. Incoordination and spasm of the arms have been noted. It is treated by lavage, saline purgatives, and cardiac and respiratory stimulants. (3) Erythema or Urticaria, with headache and malaise.

In acute attacks the rash appears in a few hours and rapidly involves the whole body. It may be very marked on the face and head, causing great swelling of the subcutaneous tissues and a striking, swollen, bloated, blotchy appearance. The child feels very ill, and often vomits or quickly develops diarrhoea. The alimentary symptoms depend greatly on the nature of the poison. It runs its course in a few hours and is rarely prolonged beyond 2 or 3 days; occasionally as many weeks. The chief complications are dyspnoea, due to serous effusion into the larynx or pharynx; albuminuria and hæmaturia; vomiting and colic. Most cases rapidly yield to treatment by an emetic, if the food has been recently taken and not vomited, saline purgatives, and bran or oatmeal baths. Sedatives are sometimes required.

*Lichen Urticatus* is a much more troublesome affection, for it may be prolonged for years in recurrent outbreaks. The appearance and



distribution of the rash are very suggestive of scabies, so careful examination must be made for cuniculi. The presence of wheals and irritability of the skin are signs of urticaria. Flattened patches are rather like lichen planus. Prurigo is a similar condition in older children, but the urticarial patches are absent and the neighbouring lymph glands are enlarged. It may be mild or severe.

Lichen urticatus is most common under 2 years of age. It may begin in the third month or even earlier. It tends to disappear in the third and fourth, but may continue up to the tenth year. It is most common in summer and in the worst cases comes out in winter as well. It is chiefly associated with gastric and intestinal troubles, rickets and teething, and may follow varicella, measles, or other infections which increase the susceptibility of the skin. It causes chronic irritation, loss of sleep, malnutrition and glandular enlargement, if there is secondary infection.

*Treatment.*—It is most difficult to cure. Change of air is sometimes needed. The garments next the skin should be of linen, silk or other unirritating material. The diet must be carefully supervised. For infants it is generally advisable to reduce the amount of milk and butter, prohibit eggs and raw fruit, reduce sugars, and give fewer meals, and more soups, vegetables and starchy foods. For older children prohibit oatmeal foods, and reduce or omit starches and cane sugar. In the worst cases an exclusive milk diet may be beneficial. As a rule it is best to give a mixed diet of fresh foods, digestible and well cooked, at regular intervals. I have not found fresh fruit deleterious and am quite convinced that it is necessary to avoid foods which are liable to set up intestinal fermentation. It is very common to find these patients taking an excess of malted foods, cane sugar or oatmeal preparations. In many instances it is best to start with a simple mixed diet and to alter it in accordance with the progress of the case. One is not justified at present in assuming that the cause is invariably the same.

The child must have a warm bath at bedtime containing bran, oatmeal, boric acid or “emollient bath powder”; or liq. carbonis detergens or ol. betulæ alb, dr. 1-8 to water 2 gallons. Soda or sulphur baths are occasionally useful. In some cases warm baths must be avoided. For the relief of itching dab on weak vinegar or eau de Cologne and water; an alkaline or calamine lotion, alone or with carbolic acid 1-2 per cent.; lead or cyllin lotion; acidi carbolic dr.  $\frac{1}{2}$ -1 in cotton seed oil oz. 6; or liq. carb. deterg., plumb. acetat., glycerin. aa dr. 2, water ad oz. 6; menthol gr. 2 ad water oz. 1; ichthyol 5, glycerin. 5, water ad 100 parts, applied twice daily. Dust freely with talc or starch powder afterwards. Ointments of ichthyol 1 per cent., b-naphthol 1-2 per cent., sulphur, resinol, tar and diachylon p.a., are sometimes beneficial. Carbolic acid preparations must be used with caution. Internal medication is primarily directed to the intestinal tract. Give an occasional dose of calomel or grey powder at night and a



morning saline draught. Or a preliminary dose of rhubarb and soda, grey powder night and morning for two days, and then salol grs. 2-3 with mucilage and aniseed, t.d.s. On the whole grey powder every other night and a mixture of rhubarb, gentian and soda, t.d.s., give as good results as most drugs. Sometimes large doses of pot. citrat. are efficacious. Wolff claims a cure in 24 hours by giving sod. phosphate, gr. 10-60, every 3 hours or after each meal, if the mischief is of gastro-intestinal origin. Ichthyol m. 1-2, in glycerine and syrup, t.d.s., or a 10 per cent. aqueous solution in doses of m. 10., is said to relieve itching and restore vasomotor tone. Bromide and chloral are required for insomnia. Quinine gr.  $1\frac{1}{2}$  for each year of age at bedtime (E. Smith), atropin and phenazone in moderately large doses (Holt), and suprarenal extract (Meachin) have been recommended. A course of intestinal antiseptics and tonics may be required. Calcium salts are of little value.

*Urticaria pigmentosa* usually begins at birth or in the first months of life as a series of attacks of nettle rash, leaving stains which fade in the course of a few months. It is either an urticaria with a sero-sanguineous exudation or an infiltration of the tissues with peculiar granular cells known as "mast" cells. Some of the cases recorded under this name are really *U. hæmorrhagica* leaving stains. In a girl, aged 18 months, the rash consisted of squamous, brownish, subcutaneous spots on the thighs, buttocks and lower part of the abdomen. They had followed an attack of varicella. At the onset the patches are dark red or reddish brown, somewhat infiltrated, the size of peas, and may cover the entire face and body. They may look like insect bites or urticaria, but they do not itch. Later on they become sepia brown or yellowish in colour. They disappear towards the twentieth year but may last longer. These children are often neurotic. Dermatographism may be present. Lime, arsenic and salicylates are the drugs usually given. Local applications are useless.

**Erythema Nodosum** (*Nodal Fever*) is an acute specific febrile disorder with an incubation period, prodromata, a stage of eruption, and a period of convalescence. It occurs in its most characteristic form in childhood. It is rare under 3 years and in extreme old age. The youngest case under my notice was 18 months old. About one-fifth of the cases occur in the first decade, two-fifths in the second, and one-fifth in the third decade of life. Under 10 years of age two females are affected to one male; and the relative preponderance of the female sex increases with advancing years, the reverse of what holds good in rheumatic fever. It is said to occur with about equal frequency in each quarter of the year and does not exhibit the autumnal rise of rheumatic fever. In my experience it is most frequent in May and comparatively rare in July and August. Three-fourths of my cases began in the first 6 months of the year. It may affect more than one in a family and sometimes assumes an epidemic prevalence. It is liable to occur in the convalescence of scarlet fever.



Its *pathology* is doubtful. The onset and course are in favour of microbial infection, but so far no organism has been recovered from the blood or the serum of artificial blisters.

Probably it is closely allied to Urticaria. At times the two diseases are present in the same patient and dermatographism may be marked. Many attacks are ushered in by gastro-intestinal disturbance, and it is likely that the disease is the result of toxic absorption from the alimentary canal or perhaps from the tonsils. Formerly it was supposed to be a sign of rheumatism and in favour of this were urged the similarity of the age incidence, the special liability of girls aged 10-15, epidemic occurrence, a suspicion of contagion, the liability to arthritis, tonsillitis, and endocarditis, and the occurrence of erythema. On the other hand it is only half as frequent as rheumatism in the first decade, and is much more frequent in females. It is least prevalent in the third quarter of the year, when acute rheumatism is most common, and is uninfluenced by salicylates. In my experience girls of 5-10 are more liable than girls of 10-15 years of age. The character of arthritis is different. No family history of rheumatism was obtained by Lenden in 63 cases and only 2 had a past history of rheumatic fever. On the other hand I obtained such a history in 10 cases out of 25 and a personal history in only 1.

*Symptoms.*—There is an indefinite prodromal period of variable severity for 1-4 weeks during which the child is ailing, dyspeptic, feverish, and suffers with headache and pains in the limbs. Sometimes there is tonsillitis or an attack of diarrhoea. One child had an attack of epistaxis on the day before the rash. The onset may be severe and accompanied by vomiting or gastro-enteritis. The temperature rises and in a day or two the rash appears. Fever may be present for some days before the rash and rarely reaches 104° F. It subsides by lysis in the second week. Occasionally it lasts for some weeks, being intermittent or irregularly remittent in character and uninfluenced by salicylates. Cases vary much in type and severity. In rare instances the constitutional symptoms are severe with much prostration, mental depression, and little rash. Or the rash may be extensive and the symptoms slight. Frequently both rash and symptoms are trivial, with no sense of illness and little pain. Delirium and coma may occur from hyperpyrexia and occasionally are due to salicylates.

The *rash* appears in 1-5 days. It is chiefly distributed on the front and sides of the shins. In more extensive cases it comes out on the knees, thighs and forearms, mainly the extensor surfaces, and on the buttocks. The characteristic nodes are raised, somewhat conical swellings, varying in colour from a bright or dusky red to a dull purple. The purplish colour is most in evidence when the limbs are cold and the rash fading. The nodes are hot, painful and exquisitely tender. Frequently only a few are present. Sometimes there is a polymorphic rash of papules, diffused blotches, and even circinate or annular patches.



Arthritis is uncommon or slight, and takes the form of pain in the joints with slight tenderness on movement but no redness and swelling, such as are seen in acute rheumatism in adults though analogous to the type of rheumatic joint affection of early life. Occasionally the joints are swollen, red and œdematous, as in gout; or may simulate the appearances of gonorrheal rheumatism or osteo-arthritis. S. Mackenzie (1897) stated that 17 out of 108 cases at all ages had arthritis and another 17 had rheumatic pains, while no less than 23 had or developed signs of endocarditis. This is quite contrary to my experience in children. Though a murmur is occasionally found, it disappears during convalescence and is no doubt due to cardiac dilatation.

The rash subsides slowly. The nodes become less hot and tender, less painful, more dusky in colour, and in late stages may be a little œdematous. The œdema of the legs quickly disappears, but will return if the child is allowed up too soon. Relapses are uncommon and recurrence rare. Occasional complications are angina, bronchitis, pleurisy, endocarditis and nephritis. Phlyctenules are quite frequent. Convalescence is somewhat slow, and characterised by weakness out of proportion to the severity of the attack and much anæmia. The prognosis is excellent. Serious and dangerous cases are associated with endocarditis, and are probably of septic origin and different in character. There is no special prevalence of cardiac affections in these children.

The *diagnosis* is easy when the rash is out. Before this the febrile state may be mistaken for influenza, gastritis, rheumatism, typhoid fever, or even meningitis if the constitutional symptoms are severe. Neither a febrile arthritis nor endocarditis is a proof of acute rheumatism, for they both occur in other diseases. A cardiac murmur is not necessarily due to endocarditis.

*Treatment.*—Keep the child in bed until the nodes have subsided and there is no fear of secondary swelling from cardiac weakness. In the erect posture the legs swell and are more painful. Apply cold compresses, lead or lead and opium lotion. Hot fomentations may afford more relief. Give a warm bath daily and keep the bowels open. Attend to the diet and digestion. During the febrile stage restrict the diet to milk and carbohydrate foods. Oranges and lemons can be given freely. Citrates, citric acid and quinine are useful drugs. Iron tonics or cod-liver oil are valuable during convalescence. For 2 or 3 weeks the heart must be watched for fear of dilatation.

**Purpura.**—*Syn. : Morbus Maculosus—Werlhoff's Disease.*—Purpuric eruptions of all kinds are symptomatic rather than special disease entities and great confusion has arisen from attempts to define separate varieties. Bleeding may take place subcutaneously, from mucous membranes or into internal organs. P. simplex, rheumatica, hæmorrhagica, fulminans and Henoch's purpura are probably the same disease varying in severity. Even



in mild cases there is usually some joint pain, swelling of the legs and intestinal disturbance. These varieties are classified by Osler as *Essential Purpura* in opposition to the *Symptomatic Purpura* of infective diseases; due to toxic causes such as drugs and snake venom; cachexia; hysteria; or of mechanical origin, as in pertussis. Such eruptions depend on altered blood states and occur in leukæmia, scurvy, hæmophilia; infective endocarditis, pyæmic affections, hæmorrhagic types of specific fevers, cerebrospinal meningitis; diphtheria, biliary toxæmia; marasmus; chronic nephritis and cardiac troubles causing renal congestion. Purpura may be associated with urticaria and angioneurotic oedema. Symptomatic purpura is of grave prognostic import though it adds nothing to the severity of the primary disease. No special treatment is needed.

*Purpura Simplex* is a simple purpuric eruption for which no cause can be found; frequently associated with no constitutional symptoms or merely with slight fever and digestive disturbance. It is mild in character and course, and ends in recovery. Anorexia, headache, nausea, apathy and general malaise may precede the eruption by a few days to 2 weeks. The rash is limited to the skin and generally appears on the extensor surfaces of the arms and legs. The face and hands often escape. The spots are variable in size and shape, bright red in colour, not confluent, and do not disappear on pressure. In size they vary from that of a pin head to large subcutaneous hæmorrhages, with the appearance and undergoing the changes of bruises. They last from 1-4 weeks and may come out in successive crops. *P. Convalescentium* is a simple purpura during convalescence from fevers such as measles, scarlet fever and diphtheria. It is very rare after diphtheria, is not due to antitoxin, and must be distinguished from hæmorrhagic diphtheria. Occasionally it is hæmorrhagic in type or fulminating and fatal.

*P. Hæmorrhagica*, the true *morbus maculosus Werlhoffii*.—The hæmorrhage is not limited to the skin; it takes place also from the mucous membranes. It is more severe and more sudden in onset than the previous variety, with a temperature of 101°-103° F., anæmia due to loss of blood, gastro-intestinal symptoms, albuminuria and oedema. The purpuric spots and subcutaneous exudations in a limb may coalesce and give it an appearance of gangrene, but without the smell. Purpuric spots may appear in the oral structures, eyes, ears, intestines and bladder. Epistaxis is the most frequent variety of hæmorrhage from the mucous membrane. Occasionally the bleeding is limited to hæmaturia. The joints usually escape. Mild cases attain a maximum in about a week and then cease, ending in recovery in 14 days. Relapses are not uncommon. Severe attacks may pass into a typhoid state and are then almost always fatal in 1-6 weeks. Rarely it is chronic and recurrent for months or years.

*P. Rheumatica*, sometimes called *peliosis rheumatica* or *Schönlein's disease*.—Both this and *P. Simplex* may be associated with other skin



affections such as urticaria, erythema nodosum and erythema multiforme exudativum. It has probably nothing whatever to do with rheumatism and is really a compound of urticaria and purpura. Thus, an 8-year old boy, after a few days illness with gastric and intestinal symptoms and probably a mild degree of fever, developed a purpuric rash on the lower limbs and buttocks, joint pains and a little swelling of one knee and ankle. He had a fair appetite and little fever. In a few days the symptoms subsided. After an interval of a few more days he suddenly developed œdema of the penis, especially the prepuce, followed by hæmorrhagic staining, and later on fresh purpuric spots and bruises. These signs cleared up in another week and then again fresh spots came out in the lower limbs.

The characteristic features of this variety are the purpura, painful joint swelling, œdema, and occasionally hæmorrhages from the mucous membranes. It is preceded by anæmia, languor, vague pains, vomiting or diarrhœa, and occasionally urticaria. The temperature rises about 2° F., perhaps 5° or 6°. The throat is sometimes inflamed. The rash is generally on the lower limbs, buttocks and forearms. It is most common on the legs up to the knees and most marked near the joints. It may be scattered all over the body, limbs, and even the face. Sometimes it resembles an urticaria with purpura in the centre of the wheals. The purpuric parts may be œdematous, secondarily to the rash, especially the legs when the patient is allowed out of bed too soon. More often the œdema precedes the purpura, or occurs independently of it in the eyelids, face, penis, scrotum, labia, etc. Joint pains are common and joint swelling is not infrequent. The ankles and knees are the most liable, and are affected with a peri-articular rather than an intra-articular effusion. Bleeding may take place from the mucous membranes, e.g., nose and bowels. Intestinal bleeding, associated with colic and vomiting, is very suggestive of Henoch's purpura. The spleen may be enlarged. The heart is not involved and there is rarely albuminuria.

Most cases recover in a fortnight. Others have relapses with rash, fever and articular swelling; and exhibit a more or less paroxysmal course, sometimes of many weeks duration but ending in recovery.

*P. Fulminans* is so called because of the sudden violent onset. It has followed immersion in cold water, sea bathing, fright, shock and scarlet fever. Von Lerber (1904) reported a case in a healthy girl, aged 13 months, who became collapsed after a restless night. An hour later bluish-red spots appeared, the pulse was quick and there was no fever. The rash extended all over, the pulse became uncountable, respirations 60 per minute, and death took place in coma 5 hours after the onset. In another case (Haw, 1903) a sturdy healthy boy, after being nearly drowned, developed epistaxis, melæna, discrete purpuric spots, insomnia, shivering, anorexia, tachycardia, pallor, debility, and splenic enlargement. The epistaxis became recurrent, splenic dulness diminished, general œdema without



albuminuria developed, and death took place from cardiac failure 3 weeks after the onset.

The usual signs are a sudden onset, chill, vomiting, prostration and high fever. A rapid and extensive eruption appears, rather symmetrical, and chiefly on the extremities. It may give them the appearance of gangrene, with bullæ but no fœtor. Delirium, stupor and coma develop. The spleen is generally enlarged and there is usually albuminuria, and less often hæmorrhage from mucous membranes.

The signs indicate an acute infection. It is usually fatal in 12-24 hours up to 4 days. Sometimes it runs a prolonged course, with relapses, and ends in recovery. Visceral hæmorrhages, e.g., into the suprarenals, are often found. In a case which occurred in a boy, 3 years old, on the eighteenth day after severe scarlatina and was fatal in 36 hours, the kidneys showed acute degeneration and were almost entirely converted into fat (Biss). This is indicative of an acute toxæmia.

*Henoch's Purpura* or *P. abdominalis* is a syndrome of symptoms rather than a special entity. It has been called by Osler *erythema exudativum multiforme* and cases have been described as purpura hæmorrhagica and angioneurotic œdema. Both sexes are equally liable. It is most common from 3-15 years of age and occurs in infants. Some of the cases of spontaneous recovery from intussusception are almost certainly of this type. The onset is sudden. It may be preceded for a few days by purpuric spots, dyspeptic symptoms or articular pains. The main feature is an abdominal crisis, characterised by severe colic, sometimes tenderness, vomiting, and hæmatemesis or melæna. The degree of pain is a fair measure of the amount of effusion into the intestinal wall and the spasm of the gut above the obstruction. The stomach contents are first vomited, then greenish mucus, and finally dark blood. Constipation is followed by diarrhœa, the passage of blood and mucus, and perhaps tenesmus. The loss of blood may be sufficient to produce marked anæmia. The tongue is coated, breath foul and appetite lost. The abdomen is retracted, hard and tender. Fever is absent, moderate or irregular. There is much prostration, phosphaturia and albuminuria. The spleen may be enlarged.

Purpuric signs may be absent. Or petechial and macular hæmorrhagic eruptions, with a tendency to symmetry, take place into the skin; about the joints, causing articular and peri-articular effusions, with pain, swelling and stiffness; and beneath the periosteum. Bleeding may occur from the stomach, bowel, nose, gums, mouth, pharynx, lungs or kidneys; rarely from the female genitals, ear, skin, and into the retina or choroid. Rheumatic pains are not uncommon and occasionally there is severe œdema. The child becomes gravely collapsed and may present the abdominal facies, dry and brown tongue, thirst and frequent vomiting, and flexion of the legs on a hard, rigid, undistended, tender abdomen. The condition is one of partial or complete obstruction, and the abdomen may become distended.



Examination reveals resistance and sometimes an indefinite local swelling. At operation there has been found hæmorrhagic œdema of the small intestine or colon. Sutherland states that in about 75 per cent. the effusion is in the ileo-cæcal region and notes that over 70 per cent. of intussusceptions are found in this situation.

Recurrent attacks occur in the same patient at intervals of days or years. They vary in severity and type, and tend to become less severe. The same child may at one time present simple purpura, at another marked arthritic phenomena, and in a third attack an acute abdominal crisis. The hæmorrhagic congestion of the bowel affects one or all the coats, and is limited or extensive in distribution. A limited affection of the small intestine closely simulates a reduced intussusception, but purpuric spots may be found in the mesentery and on the peritoneum of other portions of the gut. Superficial ulcers or extensive necrosis of the mucosa and sub-mucosa may be present. Acute nephritis has been noted and proved fatal in a few days. Endocarditis has also occurred.

The *blood* has been found sterile in the few cases in which it has been examined. Crawford (1903) noted a reduction of hæmoglobin to 68 and 77 per cent.; polynuclears 53 and 81 per cent. One case showed a leucocytosis of 60,000 and a diminution of red cells; the other, neither leucocytosis nor erythropenia. Thus the blood changes are not characteristic. In essential purpura there may be found slowing or absence of coagulation; reduction in specific gravity, hæmoglobin, and number of red cells and blood platelets; megaloblasts and microblasts, an occasional myelocyte, and leucopenia, except perhaps at the onset. Leucocytosis has been found in *p. hæmorrhagica*. The changes are much the same as in scurvy and secondary anæmia. A polynuclear leucocytosis is generally due to secondary or symptomatic purpura of infective origin.

*General Etiology of Essential Purpura.*—It may occur at all ages, especially 2-12 years, and very rarely in infancy. Both sexes are equally liable, or there is a slight preponderance of females. It is most frequent in the winter months. Bad food, malnutrition and imperfect hygiene are predisposing factors. Gastro-intestinal symptoms are so often present as to suggest that the cause is a toxæmia of alimentary origin. In a few instances the antecedent sore-throat may be the source of infection.

*Pathology.*—Some change in the blood permits extravasion. The disease is vasomotor, toxic or infective in origin. Hensch regards the abdominal variety as a vasomotor neurosis. The symptoms are due to effusion into the wall of the gut. There is no connection with rheumatism and cardiac lesions are distinctly rare. Joint pains are due to hæmorrhagic or serous effusion into or around the joint. The similarity of *p. hæmorrhagica* to scurvy must be noted; the blood picture is analogous. The disease is closely allied to urticaria, an auto-intoxication by a vasomotor toxin. The close connection of the various types is brought out by the



clinical pictures thereof, and their occurrence in the same patient. This in itself suggests a common origin, and the symptoms and anatomical changes of profound toxæmia in fatal cases may be regarded as evidence that the minor ones have a similar etiology and pathology. Minute visceral hæmorrhages in the serous membranes and omentum, sero-sanguineous effusions into various cavities and splenic enlargement, usually due to hæmorrhage, are found after death; and sometimes profound degenerative changes in the kidneys.

*Diagnosis.*—The chief affections to bear in mind are scurvy, cerebro-spinal fever, malignant infective disorders, intestinal ulceration, acute nephritis and renal calculus. Hæmaturia, without other signs, may not be recognised as purpuric. Henoch's purpura is apt to be mistaken for intussusception, acute colitis, appendicitis, peritonitis, acute intestinal obstruction or gastric ulcer. Several cases have been operated on. Distension of the abdomen increases the difficulty of diagnosis, unless some purpuric spots are found. An abdominal crisis may be due to serous effusion in angioneurotic œdema. Intussusception may occur in the course of the disease.

*Course and Prognosis.*—It may get well in a few days, continue for many months in a series of relapses at uncertain intervals, or end fatally. The abdominal type lasts a few hours or days and undergoes gradual remission. The prognosis is better in the older children. It increases in gravity with the severity of the symptoms and the signs of sepsis. No definite end to a case of medium severity can be accurately foretold. The simple variety is very rarely fatal. Complications are rare. A retro-pharyngeal blood cyst was present in a girl, aged 8 months, and fatal hæmorrhage resulted on opening it (Blaker, 1904). Sloughing or gangrene of the skin and mucous membrane may occur, and is generally a sign of fatal prognosis. In mild cases slight injury or pressure may cause sloughing. Both the uvula and prepuce have sloughed. Hæmaturia is common, so too degeneration of the renal epithelium in the tubes, but acute nephritis is rare. Chronic nephritis has been known as a sequel. Endocarditis and pericarditis are unlikely to develop, except when there is acute rheumatism or sepsis. Fatty degeneration and cardiac dilatation occur in prolonged cases. Meningeal and retinal hæmorrhages are uncommon. Convalescence is often slow.

*Treatment.*—Keep mild cases in bed, because of the risk of relapses. In severe attacks the horizontal posture is essential. Avoid exposure and injury. Give small meals of simple, nutritious, digestible food. Fresh fruit and vegetables are most beneficial. Calcium salts may increase the coagulability of the blood and adrenalin can be applied to stop local bleeding. Adrenalin and arsenic are prescribed for hæmatemesis. Keep the bowels open with calomel and use it as an intestinal antiseptic. For purpura abdominalis, with pain and hæmorrhage, give small doses of castor oil



and opium, or opium alone, an ice bag to the abdomen, and atropin gr.  $\frac{1}{200}$  sub cutem. Aromatic sulphuric acid is useful as a hæmostatic. Ergot and chloride of iron are of doubtful value. Quinine must be given in infective cases with fever and rigors. If the bleeding is long continued, recurrent and severe, gelatine should be given by the mouth. Fresh horse serum is sometimes most efficacious in this as well as in other hæmorrhagic diseases. Iron, arsenic and cod-liver oil are needed during convalescence.

**Hæmophilia.**—This tendency to bruising and bleeding is a congenital peculiarity which is transmitted through many generations by the female members and affects the males. Of the two sexes only one-twelfth are females and they are less severely affected. Females show no tendency to excessive bleeding during menstruation or parturition. They are more prolific than normal women and have a higher proportion of daughters. Transmission by males to sons and daughters has been recorded. Thus 6 out of 15 cases in one family were females and 10 of the 15 inherited through a male parent (Larrabee). The male is more likely to transmit it if he is unaffected. Otherwise he may not live to procreative age. Grandidier (1885) found 20 cases in a population of 165 in the village of Tenna, and the diathesis could be traced back to 1770, and subsequently to a single remote ancestor. Osler recorded it in 7 generations of the Appleton-Swan family; Muir in 8 generations, transmitted from a male born in 1783; and Dunn collected 770 cases in 256 families.

The disease is independent of race and climate, but more common in the temperate zones. Some cases die shortly after birth from bleeding in the first week of life. Grandidier found a family history of hæmophilia in 14 out of 228 cases of spontaneous umbilical bleeding. Including these and one of his own, Larrabee (1906) collected 37 cases of hæmophilia in the newborn, i.e., a family history of the affection or the child survived and proved a "bleeder"; males 31, females 6; 22 fatal; 24 families involved. The bleeding is commonly umbilical but may occur from the skin, mucous membranes or into joints.

**Pathology.**—Recent evidence is in favour of the view that it is due to a ferment defect in the blood and that it can be cured by injecting fresh serum. It is probably a primary blood affection rather than one of the vessels, although fatty degeneration of the intima has been found. Possibly the ovarian secretion renders females less susceptible. The blood picture is that of severe anæmia and varies with the amount of blood lost, showing decrease in hæmoglobin, poikilocytes and hæmatoblasts. The leucocytes are normal or slightly decreased in number, and there is a relative lymphocytosis. It is deficient in coagulability and in zymoplastic substance. After bleeding for some minutes the blood may coagulate much more quickly than normal.



Fatal cases may show arterial hypoplasia, a thin right ventricle and interventricular septum, hypertrophy of the left ventricle, a large thymus, and secondary arthritic changes.

*Symptoms.*—Bleeding rarely begins in infancy, sometimes in the first or second year, and only occasionally after the twenty-first. These subjects are usually thin, nervous and intellectual, with transparent skin and prominent veins. A spontaneous attack may be preceded by irritability, restlessness, lassitude, headache, vertigo, scanty micturition and constipation; and ushered in by vasomotor disturbance, viz., flushing, throbbing of the temples, and disturbances of sight and hearing, shortness of breath and pains in the limbs. The bleeding is apparently spontaneous or follows some trivial injury, e.g., strain, contusion, scratch, cut, tooth extraction, ritual circumcision or paracentesis of the tympanum. It is internal, external or arthritic. External bleeding takes the form of epistaxis in half the cases. It may occur from any mucous surface and even beneath the nails. It is a persistent oozing, liable to end in death from syncope. Or the bleeding may be subcutaneous (petechiæ, hæmatomata), intramuscular, into serous cavities or meningeal. It never affects the face. Internal hæmorrhage is variable in rapidity of development. It causes severe pain, rapid pulse and rise of temperature.

Arthritic hæmorrhage is spontaneous or due to injury. It may be the first sign and, if so, the joint is liable to be opened for supposed acute arthritis. It is rare under 7 years of age. Swelling, stiffness and slight fever, with no external discoloration, are present. In order of susceptibility come the knee, hip, elbow, ankle, wrist and shoulder. The fluid is more or less rapidly absorbed, except in the rare instances in which it persists for months and causes permanent stiffness, the result of secondary synovitis, changes in the cartilages and bone, and partial or complete ankylosis. Osteophytic changes follow recurrent attacks. An affected knee closely resembles tuberculous disease. Often there is more than one joint involved. It is treated by rest, splints and pressure. Puncture may possibly be justifiable; incision never.

*Course and Prognosis.*—Litten states that 60 per cent. die under 7, and 89 per cent. under 10 years of age. Hæmoptysis, hæmatemesis, melæna and hæmaturia are very rarely fatal. Death is uncommon in a first attack of bleeding. Recurrent attacks with arthritic symptoms, like rheumatism, are somewhat periodical in character. The severity of the bleeding varies in different children and the tendency is less as age advances. Complications are rare. Gangrene of the skin and Volkmann's contracture, after intramuscular hæmorrhage, have been recorded. The blood in internal hæmorrhage is generally reabsorbed but the clot may persist and become calcified.

*Treatment.*—The female members of hæmophilic families should not bear children. The affected children must live a quiet sedentary life, in



warm climates, play no boisterous games, take no alcohol and live on simple diet. Vaccination is not dangerous. Teeth must not be extracted.

For capillary oozing apply a pad of gauze soaked in sterilised gelatine or calcium chloride 2 per cent. solution, adrenalin 1 in 1000, or salt solution. Apply local pressure and elevate the part. The actual cautery may be necessary. Internally give large doses of calcium lactate, gelatine, adrenalin or ergotin. These are all useless (Sahli). Emile Weil (1907) recommends the subcutaneous injection of fresh rabbit's serum, to prevent or stop bleeding. It increases the coagulability of the blood. The serum of man and horse, or antidiphtheritic serum, not more than 2 weeks old, can be used, and possibly it will prove efficacious when given internally or per rectum. Thyroid and ovarian extract have also been credited with success. The subcutaneous injection of gelatine is useful but not very safe (p. 130). Morphia is given for internal bleeding, if there is much pain. Calcium salts are given at intervals to maintain blood coagulability. Arsenic, iron and dilute sulphuric acid help to preserve the general health.



# CHAPTER XLV.

## THE URINARY SYSTEM.

*The Urine — Normal and Abnormal Constituents — Albuminuria —  
Hæmaturia — Hæmoglobinuria — Micturition — Diabetes Insipidus —  
Enuresis — Affections of the Bladder.*

The Urine can be obtained in infancy by passing a No. 2 catheter ; by pressure on the bladder from above downwards after an interval of sleep ; by sitting the child on a cold pot every 15 minutes and applying cold or heat to the hypogastrium ; by means of a condom fixed to the penis, the insertion of the penis in a bottle between the thighs, or a special rubber apparatus ; by a small cup over the vulva and between the thighs ; by means of a sponge or pad of absorbent wool, or a macintosh arranged so that the urine runs into a receptacle beneath the bed ; or by giving a suppository or clyster, and collecting the urine which is rarely passed simultaneously with the fæces.

*Quantity.*—In early life the amount, specific gravity, and percentage of the different constituents vary from time to time to a much greater extent than in adults and are more readily affected by slight causes. The newborn child passes  $\frac{1}{3}$ - $\frac{3}{4}$  oz. at each evacuation. The amount varies in the first few days with the ingestion of fluid. Very little may be passed for 3 days. On the fourth day the quantity reaches 200 c.c., and on the seventh day 300 c.c. ; about two-thirds of the amount of milk taken. The following table shows marked variations :—

QUANTITY OF URINE (REUSING).

Day of Life.	No. of c.c.	Day of Life.	No. of c.c.
1	2-61	5	22-222
2	11-145	6	70-280
3	13-171	7	93-338
4	17-179	8	100-331

The quantity increases but does not correspond exactly with the fluid taken. It is greater in the bottle-fed. Proportionately to weight it is 4-5 times the amount passed by the adult. From 1 month to 2 years of age



the quantity is 200-600 c.c., an average being about 10-12 oz. Towards the end of the first year 68 c.c. of urine is equivalent to 100 gms. of nourishment (Camerer). A rough scale is 10-15 oz. in the second year and an additional 1-1½ oz. for each year of life up to the twelfth. These amounts are often exceeded.

The *specific gravity* depends on the fluid taken and the amount of urea. It is 1008-1012 during the first 3 days; falls to 1003-1004 and remains low for some months; and throughout childhood is much lower than in adults, reaching 1015-1020 in boys at 14 years.

*Reaction*.—In the newborn it is acid, high coloured, turbid, contains mucus and often deposits urates and epithelial cells. Albumin and casts may be present for some days. After a few days it is very pale, almost odourless, and neutral or faintly alkaline. In atrophy and sclerema it is acid. The presence of free alkali is recognised by holding moist litmus paper over the test tube while heating. Turbidity clears up on heating, if due to urates; and on the addition of acetic acid, if due to phosphates. If filtered urine is turbid, the cause is either bacteria or blood. Alkaline urine is due to digestion (the alkaline tide), vegetable diet, and excess of carbonates and salt. Acid sodium phosphate makes it acid. Ammoniacal urine is turbid and offensive, usually due to excess of fatty food.

*Urobilin and urobilinogen* are derived from bilirubin (p. 273). Urobilinogen is absent in the breast-fed, present in the bottle-fed, increased in intestinal affections, hæmorrhages and paroxysmal hæmoglobinuria, and absent in obstructive jaundice. It forms a colourless compound in an alkaline medium.

*Urea*.—The percentage of urea in the urine is 1·5-1·7 in children, and 2·2 in adults. More protein per unit of weight is taken by children, but much nitrogen is used up in the formation of new tissues. Per kilo of weight the amount of urea excreted at 3-6 years of age is 1 gm., at 8-11 years is 0·8 gm. (Uhle) or 0·7 gm. (Sondern), and at 13-16 years is 0·3-0·5 gm. (Sondern). About the second year there is usually an increase because more protein food is given. The amount varies directly as the supply of protein and inversely as the cell-growth. The bulk of the nitrogen is excreted as urea. Urea is diminished and the ammonia-N increased in nutritional disorders (p. 151). The percentage of urea in the urine is higher in later childhood, particularly in boys, because of increased exercise, loss of fluid by sweating, and a liberal supply of nitrogenous foods. For *lithuria*, vide p. 148.

*Purin bodies* include xanthin, hypoxanthin, adenin, guanin, thein, caffein, theobromin and uric acid. The amount excreted per kilo of body-weight varies with the amount ingested (exogenous purins) and with the extent of cleavage of nuclein in the body (endogenous purin). Idiosyncrasy, hereditary and constitutional tendencies, cold, fevers and gastric disturbance influence the amount. They are precipitated by ammoniacal



solutions of silver nitrate, and by cuprous sulphide in the presence of sodium bisulphide.

*Uric acid* is very variable in amount. It is tolerably constant during the first few days of life and allantoin, an allied body, may be present.

*Uric acid infarcts* or *concretions* are composed of uric acid, oxalate or urate of ammonia, and epithelial debris. They were present in the tubuli uriniferi of 64 out of 199 infants dead under 30 days of age, most of them in the first 3 days of life (Schlossberger), and are sometimes present in the foetus and stillborn. Henoch has noted them 7-8 weeks after birth. They form yellowish red, brownish yellow or light yellow striæ in the tubuli recti of the pyramids, converging toward the hilum. Microscopically, they consist of angular or spherical bodies in the urine, and granular masses or yellowish brown cylinders in the straight tubules. The passage of these concretions down the ureters may cause tenderness in the loins, renal colic, restlessness, retention or suppression for many hours, screaming, dysuria and even convulsions. Usually they are not sufficiently large to become impacted and cause pain, and they are found as brick-red sand or small mortar-like masses on the napkins and in the chamber pot. They rarely cause local irritation of the foreskin or vulva, and enuresis.

*Indican* is not present in the newborn or the breast-fed, except in minute quantities. It is increased by intestinal troubles, acute and chronic, typhoid fever, and constipation; and is often found in tuberculosis, chorea and acute illnesses. It is of little diagnostic value, except as a sign of intestinal derangement or the decomposition of albumin in various cavities, e.g., decomposing empyema, gangrene of the lung and bronchiectasis. It is formed from the indol in the alimentary canal and appears in the urine as indoxyl-sulphate of potassium and as a saccharate, but not as indican. Obermeyer's and Jaffé's tests are the best. Indigo-uria has been reported.

*Inorganic salts* vary much with the amount ingested. The quantity is small in the newborn and increases as age advances. The amount of chlorine depends on the intake, if the kidneys are healthy. Insoluble carbohydrates form a cloud on heating the urine of those who eat many vegetables. It is dissolved by strong nitric acid with effervescence.

Phosphates are normally present as acid phosphates of Na. and K., and earthy phosphates of Ca. and Mg. Earthy phosphates are precipitated by the addition of alkali. They are found in excess in marasmus and rickets; after much vegetable food; after violent vomiting and conditions in which HCl is deficient; dilatation and motor inefficiency of the stomach, and neurasthenia. The ratio of earthy to other phosphates in adults is as 1 : 2, and it may rise to 5 : 2. They may be deposited in the bladder, and excreted as milky fluid at the end of micturition; or as an iridescent pellicle on the surface of the urine; or on boiling unacidified urine. The crystals are stellate, rectangular plates or coffin-lid in shape according as



they consist of phosphates of calcium, magnesium, or ammonio-magnesium. *Phosphaturia* is a disturbance of phosphoric acid excretion, diminished acidity, or an excessive excretion of lime by the kidneys instead of by the intestines, the normal channel. The urine is alkaline. Its acidity is decreased in hyperchlorhydria, for less acid is available for the urine; in cystitis, because of ammoniacal decomposition; and in wasting diseases, because of disintegration of nucleo-proteins. Treatment consists in the administration of inorganic acids and nux vomica, attention to the neurasthenic state, avoidance of worry and overwork, mental and physical, and liberal diet, containing less fruit and vegetables.

*Sulphur* excretion is due to transformation of protein. Large quantities of ethyl-sulphuric acid and phenol in the urine are a sign of intestinal decomposition.

**Abnormal Constituents.**—Acetonuria has been considered (p. 149). *Ehrlich's reaction* or the *diazo-reaction* is present in late measles, typhoid fever (90 per cent. in the first week), and miliary tuberculosis; may be found in laryngeal affections, pneumonia, pleurisy, meningitis of all kinds, tuberculosis of the lungs or peritoneum, diphtheria, scarlatina and erysipelas; and is absent in pertussis, mumps, influenza, sepsis, rickets and nervous, digestive, circulatory and genito-urinary diseases. Mix the urine with an equal quantity of a concentrated solution of sulphanilic acid in weak HCl, and a few drops of sodium nitrite solution. It turns red on adding ammonia and gives a ring-shaped green deposit on standing.

*Sugar* is found as the result of diabetes and alimentary disturbance (p. 162). Lactosuria is occasionally seen in the breast-fed. Sugar occurs in the urine as: (1) Monoses; (a) Pentose; (b) Hexose, (i) glucose, (ii) levulose; (2) Bioses; lactose. It has been found by Demme in pseudo-hypertrophic muscular dystrophy, by Binet in severe diphtheria, and sometimes in diseases of an asphyxial character, e.g., pertussis. The best tests are fermentation, the phenylhydrazin test, Fehling's, Trommer's, and Bottger's bismuth test. Non-fermentable copper-reducing bodies are: (1) *Glycuronic acid*, perhaps due to camphor, turpentine or fevers; (2) *Pyrocatechin*, much increased by protein diet; (3) *Alkapton*; (4) *Kreatinin* (for test, v. Acetonuria, p. 149).

*Pentosuria* is a family anomaly of metabolism discovered by Salkowski and Jastrowitz (1892) and described by Salkowski and Blumenthal (1895). The youngest case up to the present was 15 years old. It is apparently harmless but important in that it may lead to an erroneous diagnosis of diabetes. It chiefly affects adult males, gives rise to no symptoms, and is not improved by anti-diabetic diet. It reduces Fehling's solution atypically. The colour is unchanged for 1-2 minutes and then turns suddenly greenish yellow or dirty orange. It does not ferment or rotate the polariscope, and forms pentazone crystals with the phenylhydrazin test.



*Cystinuria* is an anomaly of metabolism of protein, possibly due to the action of some organism. It may be produced from diamines but these bodies are by no means always found in the urine and fæces. Scholberg and Garrod investigated the urine of a boy, aged 12, with cystinuria and found diamines on 3 out of 10 occasions. Once putrescin was separated as a pure product ; twice it was found with a smaller portion of cadaverin. These compounds are dibenzoyl derivatives. Garcia also found putrescin in a case. The disease is often hereditary. Sometimes the patient is tuberculous or of a tuberculous stock. The cystin is in the form of calculi, single or multiple, or transparent hexagonal crystals. They are soluble in ammonia. These children are difficult to diet. It was detected in two children of the same family at 14 and 21 months of age (Abderhalden). On acidulating the urine with HCl, crystals can be skimmed off in an hour.

*Alkaptonuria* is another endogenous metabolic freak of the same type. It has been thoroughly investigated by A. E. Garrod, who groups it with cystinuria, pentosuria and albinism. These affections are harmless, unless there are secondary ill-effects. Direct transmission is rare. Several members of a family, generally collaterals of the same generation, are affected. Consanguineous marriages predispose to albinism and alkaptonuria. Garrod's conclusions (1908) are based on many collected cases :—

		Albinism.	Alkaptonuria.	Cystinuria.	Pentosuria.
Males	.. ..	34	31	63	19
Females	.. ..	28	7	30	7

Osler and Orsi have noted direct transmission ; and Garrod remarks on the special liability of first cousins (8 out of 17 families) to alkaptonuria. It is most common in males. Its appearance dates from earliest infancy, even the second day of life, and it may persist throughout life. Rarely it develops later, or temporarily during illness. It may give rise to dysuria and pollakiuria ; and to ochronosis in later life. The urine is normal when passed but becomes deep brown or black on exposure to air, staining diapers and other garments. It is darkened by alkali, reduces Fehling's solution, and gives negative results with fermentation, the polariscope and the bismuth sugar test. It contains homgentisic acid and sometimes uroleucic acid, as a by-product (Garrod).

It probably arises from tyrosin, a product of the pancreatic digestion of protein, the change taking place in the tissues. The process is arrested at an intermediate stage. It is not present until food has been taken. If tyrosin is given by mouth to these subjects the homgentisic acid in the urine is increased. A meat diet has a like effect.

*Coloured Urine.*—Eosin-coloured sweets, e.g., musk lozenges, make the urine red, so much so as to suggest hæmaturia. It has a peculiar greenish fluorescence, and is pink or green according as it is seen by transmitted or reflected light. The colour may be that of a weak solution of eosin. Methylene blue passes into the urine in 15-30 minutes. Purple or violet sweets, coloured by aniline dyes, produce blue or green urine.



Methylene blue is the common cause. In small doses it produces a greenish tint, perhaps only present in the urine secreted during sleep. The urine quickly colours white blotting paper; is decolourised by caustic potash; and readily gives up its pigment to chloroform, which becomes blue. An amyl-alcohol extract will show the absorption bands and give the chemical reactions of the dye. The urine is made green by biliverdin; olive green to black by carbolic acid, creosote, turpentine, arbutin (*uva ursi*), fuchsin in small doses, and alkapton, the colour may only develop on exposure to air; red or purplish, if alkaline, after large doses of fuchsin, rhubarb or senna, due to the chrysophanic acid in the last two drugs. Chrysophanic acid turns acid urine deep yellow or brownish yellow, becoming dull red on adding an alkali. Logwood may make alkaline urine reddish or violet. The red colour does not fade on standing and is precipitated by barium chloride. The yellowish green colour of acid urine, due to *tosantonin*, turns red on adding alkali. The red colour fades on standing and is not precipitated by barium chloride. *Purgen* causes a crimson red colour in alkaline urine, decolourised by acid. If on adding ferric chloride solution the urine becomes reddish brown or reddish purple, the *salicyl radicle* is present, provided the colour persists on boiling and is insoluble in ether. If due to diacetic acid, the colour is discharged by boiling and is soluble in ether. Antipyrin and similar drugs have the same effect as salicylates. A very dark inky colour is due to tannates or gallates. Tincture of *guaiacum* gives a greenish blue reaction, if iodides have been taken. Bile pigments turn the urine a colour varying from bright yellow to reddish brown, like beer.

*Albumose* is precipitated by acid, disappears on boiling and reappears on cooling. It has been found in measles, scarlatina, diphtheria, mumps, typhoid fever, and pyæmia; during the absorption of exudates in pneumonia and pleurisy; in blood effusions, leukæmia and purpura. The *Bence Jones albumose* is present in myelopathic albumosuria. *Peptone* may be present in pneumonia, empyema and other suppurations, malnutrition, diphtheria and nephritis. It is almost always accompanied by albumin.

**Albuminuria** is a sign of imperfect health, but by no means always serious. There may be no structural renal changes. If so, it depends on mechanical, hæmatogenous or functional causes; e.g., cardiac failure, anæmia and vasomotor disturbance. In many cases the nutrition of the renal epithelium is impaired. Albuminuria is not uncommon in acute diseases and, in conjunction with casts, is a sign of toxæmia and to a certain extent increases the gravity of the prognosis. The most delicate tests show that urine always contains albumin. Only an amount which responds to the common tests needs notice. Add a drop or two of dilute acetic acid to the urine and boil the upper half. If albumin is present, it forms a cloud of varying density which does not disappear on adding nitric acid. The next best test is the cold nitric acid one. Or drop a 1 gr. tabloid of salicyl-sulphonic



acid into a pipette and draw urine up into it. If albumin is present it becomes cloudy. It is important to distinguish albumin from nucleo-protein or nucleo-albumin. Like albumin it gives a cloud with the acetic acid test, but the precipitate clears up on adding nitric acid; and it gives no white ring with nitric acid in the cold. An excess of nucleo-protein is a sign of irritation of the urinary tract below the tubules. Thus it is found in pyuria, and if there are many crystals of oxalates, phosphates or uric acid.

*In the Newborn.*—It is variously stated that albumin is present in the urine of 40-100 per cent. of newborns, and may persist for 2 weeks. Flensburg regards it as a nucleo-protein. Ssesenowski points out that mucin is frequently mistaken for albumin. He found mucin nearly always present, albumin in 22 and uric acid in 60 per cent. The albumin is due to hyperæmia from circulatory changes at birth, or to renal irritation by highly uratic urine or renal infarcts. Possibly it is connected with incomplete development of the glomeruli, a persistent foetal state, or metabolic changes consequent on the food supply. Even if nucleo-protein is differentiated from albumin, it is true that albuminuria is quite common in the newborn and may be considerable in amount. It is of little pathological significance. Casts and renal cells are found on centrifugalisation, but even in conjunction with albumin are no proof of nephritis. A few cases of congenital or intra-uterine nephritis are on record. The possibility of chill or infection, at or shortly after birth, must be carefully eliminated before accepting such a diagnosis. Acute nephritis is liable to be overlooked in early life because it is unsuspected. On the other hand infantile œdema is by no means always due to nephritis, and indeed is rarely of renal origin.

**Functional Albuminuria.**—It is by no means uncommon to find albuminuria in apparently healthy children. It is constant or present for some part of nearly every day, disappears in the horizontal position, and is unassociated with organic disease. This was described by Moxon (1878) as “albuminuria in the apparently healthy.” It has received numerous names since, according to the various theories of its causation and the conditions under which it occurs. Thus it has been called “physiological,” a name best reserved for “latent” albuminuria, always present but only discovered by special tests; “alimentary” or “dietetic,” if due to excess of protein food, but the influence of diet is extremely small; “postural,” “orthotic” or “orthostatic,” because it depends on posture or exercise, the name orthotic implying that it occurs while at rest and orthostatic that it follows exercise; “athletic” or the “albuminuria of adolescence,” because it follows excessive exercise during which blood pressure is raised and the splanchnic veins contract, while those of the muscles dilate, a stage followed by reaction and dilatation of the splanchnic veins and renal stasis; “intermittent,” “paroxysmal,” “cyclic or cyclical,” for it is apt to occur at certain periods of the day only; “vasomotor,”



for it follows cold baths, bathing and exposure to cold, through the effects on the splanchnic area and renal stasis; "neurasthenic," for there is frequently a marked nervous element in these cases and it may follow psychical shock. It is also called early, benign, spurious, transient, essential, anephritic, and the albuminuria of puberty, youth or development. The name "Functional" is most appropriate for it indicates neither organic disease nor a special pathology.

*Etiology.*—The nomenclature gives a fair idea of the chief factors which may be concerned in its production. Often there is a neurotic ancestry, and the father may have been similarly affected. All the children of the family may suffer. Both sexes are liable, girls much more than boys. The common age is 9-14 years. Dukes found over 16 per cent. of the boys entering Rugby school at 13 years of age were affected. Statistics of children from 10-18 years of age vary from 17-22 per cent. Probably the percentage would be higher, if every specimen of the daily urine were examined. Exciting causes are insufficient sleep, malnutrition, the strain of examinations, backwardness at school and the fear of superannuation, and masturbation or sexual development by indirectly producing nerve exhaustion.

*The Urine.*—The quantity is decreased, and more is secreted by night than day. Polyuria is easily induced by extra fluids. That secreted by night may contain a mere trace or be entirely free from albumin, and that secreted for a few hours after rising may contain serum albumin, serum globulin and nucleo-protein. During the day the albumin gets less, and is scanty or absent at bedtime. The amount may be increased by food, cold bath, exertion or emotion. The percentage varies from 0.01-0.1, and may exceptionally reach 0.5, the urine going solid on boiling. It depends on the quantity of urine, being low in diuresis. There are no renal cells or casts, except a very few small hyaline ones at times. Urates, oxalates and phosphates may be in excess in the diurnal urine, and raise the specific gravity. The albuminuria may be intermittent, or continuous and varying in amount. In one case the morning urine was always free but the evening specimen contained at different times phosphates, nucleo-protein, and nucleo-protein and albumin. It is by no means uncommon in the evening specimen after over-exertion. Sometimes it is absent for days. The amount varies with the quality of the pulse, increasing as it becomes small and weak.

*Pathology.*—There is no doubt that the albuminuria has much to do with the assumption of the erect posture for it may appear within 15 minutes. It is the result of a functional disorder of the renal circulation, probably glomerular, for it is associated with a diminution in quantity, increased acidity and decreased excretion of chlorides. The change in the renal circulation may be hydrostatic and of vasomotor origin, of which there is frequently other evidence. Blushing is common at this age. It would be unjustifiable to assert that all cases are of the same type and pathology.



An unstable nervous mechanism is apparently absent in some instances. We must consider the effects of the action of gravity; diminished coagulability or lessened viscosity of the blood, due to the abstraction of lime salts during the period of active growth; and the rapid changes in blood pressure produced by cold, etc. Blood accumulates in the splanchnic area on assuming the erect posture and from constriction of the peripheral vessels.

It is not due to dietetic causes or the ingestion of food at breakfast. There is no increase after the midday meal, nor does the omission of breakfast render the urine less albuminous. It may disappear on a milk diet although the vertical posture is allowed; possibly because of the calcium in the milk. Such a diet may increase the albumin. If the child is kept recumbent during the day, the albumin will appear on getting up at night. Overfeeding and massage do not produce it when the child is kept in bed. Sitting up in bed is less provocative than getting up. Estimations of blood pressure may show a difference of mm. 30-40 in the recumbent and the erect posture. Sutherland found one or both kidneys movable in 15 out of 40 of his cases; in 10 of which pain referable to the kidney was present and relieved by recumbency. Tessier separates a group in which serum globulin is present and perhaps a dilated stomach, enteroptosis, movable kidney, mental depression, fatigue and digestive disorders; all more or less signs of neurasthenia. On the whole we must regard the bulk of the cases as due to renal vasomotor disturbance, perhaps dependent on neurasthenia and one of the symptoms of neurasthenia at puberty. Yet all are not of this type, for it would be hardly justifiable to assume that about one-fifth of all children are neurasthenic. It is quite sufficient to ascribe the condition to vasomotor instability and to regard this as of so common occurrence as to be almost a normal peculiarity of childhood. No doubt in many cases it is partly hæmatogenous.

*Symptoms.*—Dukes recognises three distinct types. The first and largest group presents *increased arterial tension*, unstable in character, and varying from hour to hour and from day to day. He ascribes it to excess of protein food, defective elimination and hereditary gout. *Deficient vasomotor tone* is the feature of the next group of children, who show cold and congested extremities, and frequent chilblains. In a third group he places the spare, highly strung, over-sensitive neurotics. The symptoms of these groups overlap.

These children are nervous, and flush or turn pale readily. Some are florid and full-blooded; or pale, flabby and ill-nourished. Others present a dull, heavy aspect, dilated pupils, and a chronic suffused condition of the cheeks, chin and forehead; cold, dusky red fingers, almost like local asphyxia, with patches of bright red, even on a hot day, but disappearing when the arm is raised. The feet are in the same state, always cold, and develop chilblains. Scattered erythematous patches may be present on



the trunk and limbs and a variable degree of pigmentation. Marie has recorded unilateral pigmentation and sweating. Œdema may be so marked about the eyelids as to suggest renal disease and a trace may be found about the ankles. Flatfoot and lateral curvature result from weakness of muscles and ligaments.

The pulse rate is quickened and varies much under examination. It may be of low tension and the heart small. Or the blood pressure is high, the heart large, dilated and hypertrophied, and palpitations present. The impulse of the right ventricle is exaggerated. The increased cardiac impulse against the chest wall is generally due to the right ventricle, and it is only occasionally that the left ventricle is hypertrophied. The signs of an atonic heart are not apparent on examination before rising in the morning. Abnormally visible and palpable pulsation may be present in the larger arteries and the abdominal aorta. Occasionally there is asthenopia, muscular twitchings and rheumatic pains.

The subjective symptoms are debility, languor, disinclination for games and mental exertion, headaches, irritable and variable temper, depression and tendency to weep for trivial causes, anæmia, pallor, attacks of faintness, flushings, palpitations, restlessness, night terrors and somnambulism. The child is always tired, even on waking, and is disinclined to get up. Epistaxis is not uncommon. Albuminuria is always present in children who faint during drill or morning chapel. The appetite is capricious, sometimes ravenous.

*Diagnosis.*—Examine the urine secreted at different times of the day. Examine for slight renal changes, pus cells and urinary crystals, spermatozoa in excess, vasomotor symptoms, and undue mobility of the kidneys. Remember that in the convalescent stage of acute nephritis and in chronic interstitial nephritis the albuminuria may be of the postural variety. According to Wright and Ross the administration of calcium salts increases the percentage of albumin if there is nephritis, but not otherwise. Wright maintains that it is a serous exudate, analogous to urticaria, and dependent on diminished coagulability of the blood; and that it can be cured by calcium lactate. This is not confirmed by practical experience. The age of the child, the variable appearance of the albumin, and vasomotor disturbances are the chief factors on which a diagnosis is based. Cases, in which the blood pressure is high and the arteries thickened, must be kept under observation for at least a year before pronouncing them absolutely free from renal disease. The significance of albuminuria depends on its causation and not on the amount. Hyaline casts cannot be regarded as of much importance if found on centrifugalisation, for they are present in many nervous and febrile states. The other microscopical signs of organic renal disease must be excluded. Digestive disturbances may cause albuminuria by the action of toxins on the renal epithelium. So, too, all toxic conditions, many drugs, and affections of the urinary and generative



organs (post-renal albuminuria). Malnutrition of the renal epithelium, permitting transudation, altered states of the blood, and organic diseases of the circulatory system must be excluded.

*Prognosis.*—Patients always recover, but no term can be put to the duration beyond stating that it is rare after 25 years of age. It usually disappears after puberty. This affords some support to the view that it is partly dependent on incomplete development of the kidneys and that they are not perfectly formed until puberty. There is no post mortem evidence that it causes future renal mischief. Nor does scarlatina make it worse; it remains absent while the patient is in bed. Clearly the prognosis depends on the exclusion of possible renal mischief as the cause of the albuminuria. Frequent microscopical examination of the urine is essential for this purpose. The intermittent character and the small amount present are features sometimes of kidney disease.

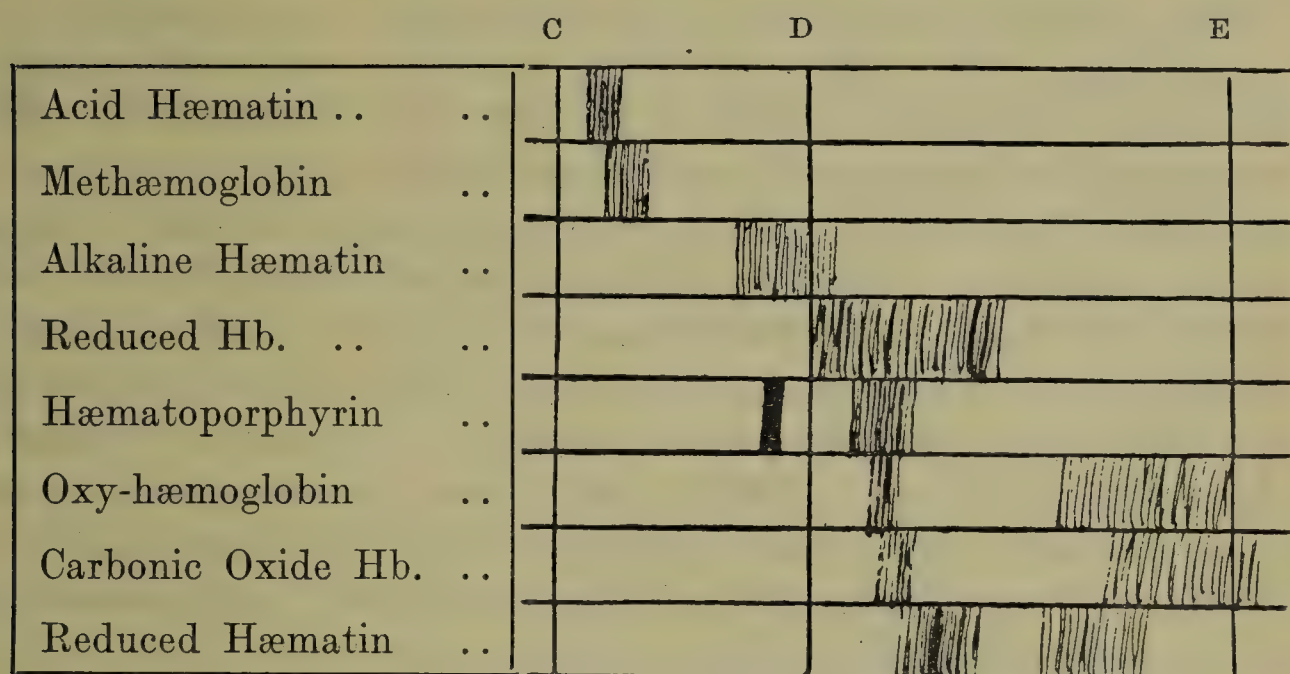
*Treatment.*—It is essential that the child should not be brought up as an invalid and that the albuminuria be not allowed to interfere with education, school life, games or a liberal food supply. Plenty of sleep, fresh air, and moderate mental work are needed. Exercise should be of the nature of recreation, rather than dull monotonous walks, but it should stop short of fatigue. Mild cases are treated on the above principles. If there is marked neurasthenia and the albuminuria is merely one symptom, the child must be relieved from all worries and causes of anxiety, lessons, and the society of other children or anxious parents. If there is much cardiac weakness prescribe isolation, liberal diet, massage, and rest in bed in a nursing home for 3-4 weeks; followed by residence in the country or a convalescent home, with recumbency from 12-2 p.m., and for 12 hours at night. Highly-seasoned food and alcohol must be prohibited. Give an extra pint or two of milk daily. Keep the bowels open. Put on an abdominal belt, if the kidney is mobile and causes pain. Drugs are rarely needed. Phenacetin relieves the headaches of the high tension cases. Bromides are useful for sleeplessness; digitalis, if the heart is weak; and calcium salts to increase the coagulability of the blood. Nux vomica and strychnia are the best tonics; followed by arsenic, iron and cod-liver oil. The treatment is that appropriate to neurasthenia in one type of case; and eliminative in the high tension variety, with reduction in the amount of protein food, meat extracts and stimulants. Once the diagnosis is established, the treatment is directed to the general state of the patient's health rather than to the albuminuria. The amount of albumin lost cannot be regarded as a serious drain on the system.

**Hæmaturia and allied conditions.**—The appearance of urine which contains blood or blood pigment varies with the amount, site of origin and nature of the colouring matter. In young infants the napkins are stained. The colour is not a reliable sign for urine may be "as red as blood" from excess of lithates, eosin, etc. (p. 550). A small amount of blood from the



kidneys is uniformly mixed with the urine, which is acid and has a "smoky" tint. The colour is due to a mixture of urinary pigments, oxy-hæmoglobin, methæmoglobin and perhaps acid hæmatin. In considerable renal hæmorrhage the bloody urine may be seen by the cystoscope coming from the ureteral orifices in jerks, somewhat like puffs of smoke. Dark porter-coloured urine is due to hæmoglobinuria or renal infarcts. Blood from the bladder is likely to contain clots. It is generally uniformly mixed with the urine, but may be passed only at the end of micturition. Blood from the urethra is bright red and passed first. The colour is more likely to be dark in renal and bright red in vesical bleeding, but there are many exceptions. Longish cylindrical clots are probably ureteral. Renal casts are a sign of renal bleeding, especially if there are many casts with adherent red cells. Microscopical examination of the sediment may reveal red cells. The spectroscope gives reliable evidence of the nature of the pigment, viz., oxy-hæmoglobin, reduced hæmoglobin, methæmoglobin, hæmatin or hæmatoporphyrin.

DIAGRAM OF BLOOD SPECTRA.



Four of these spectra possess one and four have two characteristic absorption bands. Methæmoglobin and acid hæmatin have accessory bands not here represented. The bands of oxy-hæmoglobin and CO. hæmoglobin are practically the same, and carmine gives an almost identical spectrum. The addition of ammonium sulphide converts oxy-hæmoglobin into reduced hæmoglobin. The band of acid hæmatin is readily converted into that of reduced hæmatin and can thus be distinguished from that of methæmoglobin, which it closely resembles.

The next most reliable test is the formation of hæmin crystals by boiling a little of the dry sediment on a cover slip with salt and acetic acid. The ordinary clinical test with tr. guiaci and ozonic ether produces a blue colour but a like reaction may be due to other substances. On adding an



alkali the phosphates are precipitated and carry down with them the blood pigment.

*Hæmatoporphyrinuria*.—The urine is pink, red, port wine, brownish or black, and contains iron-free hæmoglobin. It is rare in children and almost peculiar to women; due to sulphonal, trional or tetronal. It has been found in rheumatic fever, peritonitis, hepatic cirrhosis and Addison's disease. Munro (1908) reported a paroxysmal case with periodic vomiting and acetonuria, in a boy, 7 years old. The pigment is precipitated by barium chloride and hydrate and extracted by acidified alcohol.

**Hæmoglobinuria**.—The urine contains blood pigment and merely a few red cells. It varies in colour from red to black. The redder it is, the more oxy-hæmoglobin it contains; the browner it is, the more methæmoglobin and often hæmatin also. The methæmoglobin is due to the action of the kidney cells, rather than to the acidity of the urine. There is usually a little albumin; on boiling, a brown coagulum of coagulated globin; and on microscopical examination, crystals of oxy-hæmoglobin, brown granular masses, hyaline and granular casts, and often oxalate crystals. *Epidemic hæmoglobinuria* has been described (p. 126).

*Paroxysmal Hæmoglobinuria* is of obscure origin. It has been ascribed to cold, congenital syphilis, oxaluria, paludism, uric acid diathesis, traumatism, over-exertion and febrile catarrh. Some cases are associated with Raynaud's disease (p. 454). It is not common in children but may begin in the fourth year. Usually the history is one of chill or slight malaise; perhaps a feeling of chilliness, vomiting and headache, or an attack of abdominal pain and screaming. In a few hours the characteristic urine is passed with pain, and persists for 12 hours to 3-4 days, gradually getting less. Slight albuminuria may continue for still longer. There may be considerable fever, up to 104° F., and icterus in severe cases. Oxalic acid crystals are usually found, possibly accidental and not causative; and vasomotor symptoms, such as pallor, slight lividity of lips and ears, and cold extremities.

The duration is variable. Many recover after puberty but others persist. In one boy it followed a chill after a hot bath, and continued weekly for more than 6 months, after which he was lost sight of. The attacks came on at night, sometimes preceded by shivering, and the urine was clear by morning. Warmth is necessary during the attacks.

The *symptomatic* variety is associated with a definite cause; usually a blood infection or a poison. The chief blood infections are pyæmia, typhoid, erysipelas, typhus, scarlatina, measles and malaria. It has occurred in severe burns, scurvy, fat embolism, jaundice, acute nephritis and the presence of nematoid worms. The poisons are chlorate of potash, arsenical compounds, phosphorus, oxalic acid, carbolic and pyrogallie acid, mineral acids, and the poison of the edible mushroom (*helvella esculenta*). It can be induced by the intravenous injection of glycerine, bile salts, distilled



water, and the blood of another animal. Some cases are due to oxaluria, or the eating of rhubarb, spinach and fruits rich in oxalates.

*Pathology.*—Possibly there is a predisposing cause, such as congenital syphilis which is present in about one-third of the paroxysmal cases. The separation of the colouring matter takes place outside the circulation by the action of the renal cells, or more probably within the circulation by the action of an exogenous or endogenous toxin, or the production of an autolysin by cold. The bulk of the disintegrated cells is passed within a few hours of the onset. Probably there is a hæmolysin in the blood serum which acts, or is formed, when the body temperature is reduced; or the serum normally contains an anti-hæmolysin which is destroyed by cold. Eason's researches (1905) are in favour of the view that the hæmolysin is normally present and that a secondary warm stage, following on exposure to cold, is necessary to cause hæmolysis. Against this it may be pointed out that if the blood is kept fluid by the addition of potassium oxalate, hæmolysis occurs on cooling. Cold and fatigue induce paroxysms. It is a curious fact that these patients actually feel colder, instead of warmer, on exertion. Uric acid excretion and the percentage of urea-N. in relation to total nitrogen are increased during an attack. Hæmolysis is a normal process, controlled by the nervous system, and some cases may be of nervous origin. In the symptomatic varieties it can apparently be brought about by different poisons.

*Oxaluria* is a cause of hæmaturia and hæmoglobinuria. It is present in paroxysmal hæmoglobinuria and in Raynaud's disease. Hæmaturia is due to mechanical irritation and hæmoglobinuria to hæmolysis by the action of oxalic acid. Noel Paton asserts that urea excretion is increased when there is corpuscular disintegration in the general circulation. As the urea excretion is not increased the hæmoglobinuria is not due to this cause, and the separation of the pigment must be by the renal cells.

Oxalic acid is endogenous or exogenous in origin. It is contained in spinach, rhubarb, sorrel, to a less extent in tomatoes, strawberries, potato, cabbage, and in tea, coffee and cocoa. Baldwin's oxalate-free diet consists of milk, meat, eggs, wheat, flour, rice, biscuits, sugar and butter. The endogenous acid is probably derived from carbohydrates. It can be formed by nitric acid from sugar, possibly from starches, and outside the body from many articles of diet. As a metabolic disorder it may be neglected, for in practice it is found to depend on the amount ingested and the acidity of the stomach contents, rendering the crystals soluble. The acid is present in the urine as calcium oxalate. Its precipitation depends on excess of oxalic acid and perhaps on deficiency of magnesium. Meat diet encourages its deposition by increasing the acidity of the urine. The crystals may be deposited in neutral, and even alkaline urine; or by the addition of alcohol. They are octohedral in shape. "Dumb-bells" are crystals of calcium carbonate and their deposition commonly depends on an excess of oxalic acid in the urine.



The *symptoms* are a low state of health, depression, headache, pains in the back, and dyspepsia ; painful micturition, intermittent albuminuria, hæmaturia and crystals in the urine. Enuresis, excess of nucleo-protein, and calculus are sequels.

*Treatment*.—Avoid worry, over-exertion and mental fatigue. Give a diet poor in oxalates, lemons, plenty of fluid, and little sugar. Prescribe pot. citrat., as a diuretic and to combine with the calcium, or sod. bicarb.; and magnesia or acid sodium phosphate to render the oxalates more soluble. Peas contain much magnesia and little oxalate.

**Hæmaturia**.—In the newborn the blood may be due to uric acid infarcts in the kidneys, congestion from asphyxia during labour, acute nephritis, traumatism at birth, or hæmorrhagic disease (p. 127). In older children it depends on :—I. General diseases : scurvy, purpura, leukæmia, hæmophilia, and malignant exanthems. II. Local affections of the *kidney*, e.g., passive congestion from thrombosis or obstructive heart disease, acute and chronic nephritis, irritant drugs, infarcts, injury, new growths, tuberculosis, calculus and bilharzia ; of the *ureter*, as by the passage of a calculus ; of the *bladder*, as in cystitis, injury and foreign bodies, new growths, tuberculosis, calculus and bilharzia ; of the *prostate*, from inflammation, ulceration and stone ; of the *urethra*, from inflammation, passage of calculi and foreign bodies, injury and accidents.

*Idiopathic Hæmaturia*, congenital, hereditary and familial, is of rare occurrence. W. H. Attlee reported three cases of recurrent hæmaturia in one family, all females. The father died, aged 30, from uræmia. Guthrie has recorded 12 cases in 2 generations of a family ; 5 males, 7 females. The females transmitted the disease and all the children showed it shortly after birth. It is not a hæmoglobinuria. Red cells and blood casts are found. It is persistent for years, variable in extent, apt to be recurrent, paroxysmal, or continuous with exacerbations. In one case it was continuous for 2 years. Exacerbations are attributed to “catching cold” and accompanied by slight fever, headache, malaise, vomiting, and pain in the back and limbs. They may depend on diet. The attacks last a few days or weeks, occur at varying intervals, are uninfluenced by posture, and become less frequent after the tenth year. No œdema, ascites or cardio-vascular changes are present ; and no excess of urea, uric acid or oxalates. Albumin may be abundant and in excess of the blood. The cause is unknown. Possibly it is a renal epistaxis or an angioneurosis, a defect of the vasomotor system.

*Renal Epistaxis* is a name given by Gull to cases of hæmaturia in the young, paroxysmal, recurrent, and persistent for many years without symptoms or a discoverable cause. They have been ascribed to a weakness of the renal capillaries and increased blood pressure. Isolated attacks in infants are probably scorbutic. Others may depend on renal congestion occurring in the course of rheumatism, influenza, glandular fever and pertussis. Such congestion can occur without nephritis. Inflammation is



probably present, if the albumin persists after the hæmaturia ceases. In rheumatic hæmaturia there are no symptoms, except pain in the loins which is followed by copious bleeding. It may follow tonsillitis. The treatment of these varieties of bleeding is by rest in bed, warmth and light diet.

**Micturition.**—Urine is not passed for some hours after birth, perhaps not for 3 days, although there is no definite morbid cause. The delay is due to deficient secretion. A renal insufficiency has been described in the newborn. The amount varies in different infants. Nurslings micturate 6-10 times in 24 hours, usually after being fed or on waking from sleep. They may pass water every hour, or more often while awake, and retain it for 2-6 hours during sleep. Bottle-fed infants may wet themselves half-hourly. The duration of the intervals increases as age advances. Under 5 years of age the child micturates from 6-7 times; from 5-10 years, 5-6 times; and after that 3-4 times daily.

*Anuria* occurs in uræmia, fatal diphtheria, and from congenital causes. *Oliguria* is present in febrile states, watery diarrhœa, some forms of nephritis, hysteria, and certain cardiac affections. *Polyuria* is found in diabetes insipidus, diabetes mellitus, chronic interstitial nephritis, hysteria, during the absorption of dropsical effusions in nephritis, digestive troubles, emotional disturbance, pneumonic crises, and as the effect of cold, the ingestion of much fluid (polydipsia), barley water, excess of sugar in the diet, diuretics and digitalis, etc. Polyuria may cause enuresis but enuresis does not always indicate polyuria. *Dysuria* depends on high acidity of the urine in fevers, sweating, hot weather, insufficient fluid ingesta, etc.; and on local irritation about the foreskin, glans penis, clitoris, nymphæ and vulva. It may be due to urethritis, foreign bodies in the urethra, and vesical or renal affections.

*Incontinence* is normal during the early months of life. Reflex excitability is great, sphincter resistance small, and cerebral control wanting. Control is acquired much earlier by some children than by others, as the result of careful training; perhaps as early as the fourth month of life. Control is established after teething begins, and at night by 1½-2 years of age. Delay in acquiring control is due to bad training, a persistence of the infantile state, backwardness or imbecility. Incontinence may depend on genital deformities, urethral irritation, threadworms, and vesical affections, and occurs in certain nervous diseases, e.g., convulsions and epilepsy.

*Retention* in the newborn may be due to inspissated mucus; in infancy, to impacted urethral calculus. It may depend on painful micturition or congenital defects, and occurs during hysteria, meningitis, enteritis, stupor and coma. Other causes are foreign bodies in the urethra, ligature round the penis, paraphimosis, and even gonorrhœal stricture (at 2½ years, Abbé). It must be distinguished from anuria. I have seen it in influenza. It gives rise to inconvenience and uneasiness, passing on into agony. Overflow incontinence may ensue. The urine must be drawn off very slowly to prevent collapse.



**Diabetes Insipidus.**—Polyuria must be distinguished from excessive frequency of micturition and diabetes insipidus from polyuria. This is a rare disease in early life, but it may date from infancy. A marked hereditary tendency is present. In the family of Peter Schwartz, born 1772, investigated by Weil, out of 70 members 21 were certainly and 13 doubtfully affected. Cases have followed injury, e.g., a blow on the left temple and fracture of the base of the skull, fright, sunstroke, specific fevers and cerebral tumour. In many no cause can be found. Schiff produced it by section of the bulb or cerebellar peduncles. The onset is usually abrupt. The amount of urine varies from 5-20 pints; specific gravity 1001-1006. It is pale in colour and contains neither albumin nor sugar. Inosite has been found in a few cases.

The patient is generally thin and neurotic, with a dry mouth and unduly red tongue, a harsh, dry, pigmented skin, thirst, frequent micturition and enuresis. The appetite is sometimes increased, more often diminished. Languor, headache, irritability and lumbar pains are not infrequent.

The prognosis depends on the cause, duration, and the presence of organic disease. It is best when the polyuria follows injury or fevers, and worst when there is a strong family predisposition. Temporary improvement and periodical relapses are common. A few recover spontaneously. Some of the reported cases are probably instances of chronic interstitial nephritis, a disease which must always be excluded before making a diagnosis. Death is due to intercurrent disease.

The pathology is uncertain. Probably it depends on an inherited functional defect in the kidneys, rendering them unable to excrete urine above a certain degree of concentration, much below what is normal. Consequently an excess of fluid must be ingested. An increase in the salts ingested causes increased polyuria, for the specific gravity of the urine remains the same. According to Meyers (1908) the amount of urine varies directly as the amount of sodium chloride and protein ingested; and the defect is one of the mechanism responsible for concentration, probably a fault in the floor of the fourth ventricle.

Treatment does good in some cases and seems absolutely inert in others. Undue restriction of fluids impairs the appetite and digestion, increases the irritability and malnutrition, and causes symptoms like uræmia. The diet should be simple and nutritious. Neither excess nor reduction of proteins has in my cases proved beneficial. Nor has a salt free diet proved of any value. General hygiene must be attended to. Valerianates, phenazone, bromides, arsenic, atropine and belladonna are the most useful drugs. Phenazone reduces the excretion of urine and the reflex irritability of the cord. Occasionally ergot, phosphorus, opium, and nitric acid have been followed by improvement. Cold douching to the spine and faradism of the lumbar region, or galvanism of the spine, may be tried if other measures fail.



**Enuresis.**—The involuntary, uncontrolled evacuation of the bladder, called “incontinence,” is normal in infancy and a common complaint in infants and children up to 10 years of age. It is an unpleasant affection, difficult to deal with, troublesome, and interferes with school life and occupations.

Certain cases depend on malformations of the bladder or urethra. In spina bifida occulta it may be due to paralysis of the pudic nerves supplying the sphincters of the bladder and rectum, and associated with faecal incontinence. *Enuresis uretica* is a rare variety, usually in girls. The ureter opens on the outer surface of the body and the urine trickles away drop by drop. The child is always wet, even immediately after micturition. The urine comes from the vulva or the urethra, near its orifice, and the opening is detected with difficulty.

A second group of cases depends on inflammatory affections. In the third group may be placed those of nervous origin. The nerve supply of the bladder consists of a cerebral centre, which receives afferent impulses from all sources and sends out efferent ones, inhibiting the sphincter or exciting the detrusor; a lumbar centre in the third and fourth segments of the lumbar enlargement, receiving afferent impulses from the bladder and sending out efferent ones by the third and fourth sacral nerves to the bases and neck of the bladder, and, therefore, to the sphincter; a sacral centre which receives afferent impulses from the bladder and distributes efferent ones to the detrusor and sympathetic nerves.

Cerebral control is in abeyance during epilepsy, deep sleep, stupor and coma, pavor and bad dreams. It may be defective on account of heredity, imperfect mental or cerebral development, or acquired as the result of bad training, anæmia, hyperæmia, malnutrition, auto-intoxication, specific fevers and various neuroses. Lesions in the spinal centres and tracts cause general incontinence. Frequently there is another factor in the shape of some form of reflex irritation :—I. Penile and vulvar affections; phimosis, balanitis, vulvitis, caruncle, onanism, urethritis, hypospadias and threadworms. II. Bladder disorders; cystitis, tuberculosis, calculus. III. Urinary conditions; polyuria, bacteriuria, phosphaturia, oxaluria, excess of uric acid, and hyperacidity. IV. Renal diseases occasionally. V. Perineal and anal irritation; eczema ani, fissure, polypus and threadworms. VI. General reflex irritation; e.g. cold bed. Not much importance need be attached to a small bladder, muscular weakness, a poorly developed sphincter or a congenitally wide urethra. Thus there may be nervous instability, inherited or acquired, of the higher or lower nerve centres, together with defective co-ordination, reflex irritation or direct bladder irritation. Sometimes the lumbar centre is unduly excitable (spinal hyperæsthesia). In such cases and in those dependent on reflex irritability the enuresis is associated with pollakiuria. In atony of the sphincter of the bladder the enuresis is continuous, the urine dripping away.



*Enuresis* might be reserved as the name for incontinence of purely functional causation, reserving "incontinence" for those cases in which a local cause is present. *E. nocturna* is the most frequent variety, about half the cases, and is usually associated with dreaming of the act. In *E. diurna et nocturna* there is intermittent incontinence, asleep and awake, and the desire to pass water must be at once gratified. *E. diurna* alone is uncommon. *E. continua* or *sine intermissione* is a rare type, generally due to some pathological state, e.g., Pott's disease. Occasionally the enuresis is associated with fæcal incontinence and sometimes is due to vesical spasm.

*Etiology.*—There is often a neurotic or tuberculous family history. The affection is transmitted from one or other parent, more commonly the mother, in many cases. The child is often of the neurasthenic type. It is most common among the poor from neglect of proper training; in cold weather, from October to March; and affects both sexes about equally. It dates from birth in two-fifths of the cases and in one-tenth begins at 1-2 years of age. Many cases are clearly a "persistence of the infantile condition." There may be a congenital weakness of the sphincter and undue strength of the detrusor muscles, undue excitability of the lumbar centres, or a lack of acquirement of the inhibitory function by the higher nerve centres. Incontinence may occur during emotion, fright and uncontrollable laughter, under similar conditions of pathogenesis. A congenital weakness of the posterior part of the urethra may explain some cases which show traces of bladder irritability after sexual development. These patients are more subject to nocturnal emissions, can bear less sexual strain, have to empty the bladder frequently, and have difficulty in retaining water when they feel the desire to micturate. Enuresis often leaves behind it a liability to recurrence on sexual or alcoholic excess in later life. Oberländer has pointed out that in the young male the *caput gallaginis* is very prominent. It is engorged by onanism, sexual excess, alcohol and highly spiced foods, and then reflexly stimulates the lumbar centres and causes irritability of the bladder. An enuresis which has ceased for years before puberty may recur at the establishment of the sexual function and be induced by onanism. Occasionally it is due to late meals, too much bed clothing, stimulants, sleeping on the back, etc., causes which produce emissions in later life. The importance of adenoids is over-rated. They are one cause of deep sleep and CO<sub>2</sub> poisoning, and act as the final straw. The removal of adenoids and enlarged tonsils has been followed by recovery, and by the appearance of enuresis in a child previously unaffected. In a few instances there is a mental and physical state which is improved by thyroid extract.

*Symptoms.*—The worst cases can be recognised by a uriniferous odour about the child from the constant wetting. Shyness, timidity and reserve may be present. Boys are sometimes irritable and morose, especially if onanists. These children may be normal in health, mentally active and



physically weak, ill-nourished, or below the average in mental capacity. They are usually nervous and excitable and reveal other signs of nervous instability, such as habit spasm, stuttering, headaches, pavor, lenteric diarrhoea, or fæcal incontinence. Acquired cases are most common in strong, full-blooded, well nourished children, 5-8 years old, with hyperacid urine. This is the acid type of enuresis. In the more common type the urine is neutral or faintly alkaline, of low specific gravity, and perhaps contains triple phosphates or oxalates, a few pus cells and a trace of albumin. The quantity is increased.

*Diagnosis.*—Whether the affection is congenital or acquired is ascertained by a few simple questions. Diseases causing polyuria and pollakiuria must be excluded. The enuresis of epilepsy is nocturnal and spasmodic, with prolonged intervals, and there may be other indications of the fit. Local sources of irritation may be found and a history of onanism obtained. If there is chronic irritation of the prostatic urethra, the urine shows a slight haze by transmitted light and a deposit of mucus and leucocytes on standing.

*Prognosis.*—The outlook is better in boys than girls. The nocturnal variety is the most curable and children with diurnal enuresis also do well. The longer the affection has persisted, especially in girls, the more difficult is it to cure. If treatment is steadily persisted in, about 75 per cent. are curable in 3 months, and almost all within 2 years. In many instances spontaneous cure takes place at or before puberty, when the genital system is fully developed, and often earlier. After puberty it is very intractable. It may cease on marriage. It is less likely to persist after puberty in boys because of prostatic development. Peyer asserts that enuresis augurs ill for future virility, and that the patients usually have less sexual desire and can bear less sexual strain. Probably he ascribes to the enuresis effects which should be put down to the onanism giving rise to it.

*Treatment.*—The mere psychical effect of change of surroundings may be sufficient to break the habit. Remedy any local affection which may be a source of irritation of the general health, the nervous system, or of reflex excitability. These affections are rarely the primary cause and constantly exist without enuresis. Carefully examine the urine for abnormalities. Bacteriuria may exist without cystitis and is most common in girls. No barbarous methods such as corporal punishment, dark cupboards, and bread and water diet, are permissible. They are cruel and apt to make a nervous child worse. Occasionally they do good. In the same category may be placed the application of nitrate of silver to the neck of the bladder, massage of the prostate and neck of the bladder, electrical applications, blistering, and epidural injections of normal saline or 1 per cent. stovain solution, the effects, if any, being psychical. C. A. Ball (1908) recommends circumcision; the passage of a large steel bougie; and cauterisation of the sphincteric region with silver nitrate under anæsthesia, to set up local



irritation and induce powerful contraction of the sphincter when urine trickles on to it. Such measures may be perhaps permissible in intractable cases after puberty. So, too, gradual dilatation of the bladder may be then tried, for in girls the constant enuresis may lead to the bladder becoming unduly small.

Cold bathing and a cold douche to the lumbar region at bedtime stimulate the nervous system and have a psychical effect. Treat the general health and promote elimination. The child should be taught to defæcate at night rather than in the morning. Fluid ingesta should be limited, and not allowed after 4 p.m., unless the urine is highly acid. The bedroom must be well ventilated, the clothing light and not too warm, the lower end of the bed raised, and a cotton reel fixed with tapes so that it rests on the spine and prevents the child sleeping on the back. Wake the child to pass water an hour after going to sleep. The diet must be simple, digestible, and suitable to the physiological needs and digestive capacity of the child. An excess of sugars and starches is often injurious by setting up carbohydrate dyspepsia. If the urine is highly acid, reduce protein food and give plenty of water and citrate of potash. In other cases it is generally advisable to increase proteins. Alcohol, tea, coffee, beef tea, highly seasoned, irritant and diuretic foods should be omitted.

In the purely nervous cases atropine, belladonna and phenazone give the best results. Bromides are useful if there is irritability of the lumbar centre. Atropine and belladonna must be pushed to the limit of tolerance, if a good result is to be obtained. Children stand these drugs well. Large doses cause flushing of the face, dilatation of the pupils, dimness of vision, dryness of the tongue and fauces, and occasionally an erythematous rash and delirium. The last two symptoms are signs that the dose must be reduced. Belladonna is said to contain hyoscyamine and to be safer than atropine, but the amount of the alkaloid cannot be as accurately measured. Give it after tea and before going to bed. Begin with m. 5 and increase each dose by m.  $2\frac{1}{2}$ , every fifth day up to a maximum of m. 20. Continue the maximum dose for two weeks and reduce it in the same way as it was increased. One grain of the sulphate of atropine in water oz. 1 forms a solution of a strength of about 1 in 500. Give it at 4 and 7 p.m., and increase one of the doses alternately, every other day, until the child is taking two doses of the same number of minims as it is years of age. D. Macalister recommends liq. atropinæ m. 90, liq. strychninæ m. 45, syr. aurantii ad. oz. 1; 5 drops are given at 9 p.m. and the dose increased by 5 drops every fourth night up to a maximum of 30 drops, or even 60 drops. It is then reduced by 10 drops every fourth night. Seeing that 1 m. of the liquor is equivalent to 20 of tr. belladonnæ, such large doses must be used with caution. The tincture is sometimes given in doses up to dr. 1, t.d.s., combined with citrate of potash and cold douching. The maximum dose is given for a month and then gradually reduced. Tr. lycopodii can be added



in similar quantities. The addition of nux vomica or bromides may render it more efficacious. Phenazone in large doses at 7 and 9 p.m. for a week, and repeated after a week's intermission, cures some cases. The tincture and fluid extract of rhus aromaticus, m. 10-30, is useful for vesical atony, but not so much so as arseniate of strychnia. Alkalies, oxide and valerianate of zinc, and triple valerianates have also proved beneficial. Thus the nature of the drug treatment shows that cases are by no means always of the same pathogenesis, and that the best results will be obtained when the causation is ascertained and the remedies suitable are then selected. It is futile to treat incontinence dependent on bladder or urinary conditions by atropin, etc., and to give strychnia when the irritability of the lumbar centres is unduly high. Each case must be treated on its merits. An irritable bladder can be educated by keeping the child in bed and lengthening the intervals between micturition. The child should not be sent to a boarding school because of ridicule, worry and mental strain.

**The Bladder.**—In infancy the bladder is an oblong, acuminate organ, outside the pelvis, with its base at the symphysis pubes. It is small and consequently micturition is frequent. Its anterior aspect is in contact with the abdominal wall over a triangular area, not covered by peritoneum. It is rarely absent, duplicated, or divided by incomplete septa into two or more compartments. Umbilical fistula, due to a persistent urachus, and cystic dilatation of the urachus have been described (p. 120). Ectopia vesicæ is most frequent in males and associated with other deformities. It is described in surgical works. A dilated bladder may be found in a foetus with imperforate urethra. *Congenital diverticulum* is rare. It has been found behind the trigone (Tillmann), arising from the lower and back part of the bladder (C. P. Johnson), and somewhat similar in situation but associated with a third ureter (Brach). Wilkinson reported a case in a girl, aged 7 years, in whom a large diverticulum communicated with the bladder by a round orifice, just external to the opening of the right ureter. It was full of ammoniacal pus.

*Inversion* of the bladder is complete or incomplete. Gross collected 7 complete cases; 5 girls between 14 months and 4 years, and 2 adult women. In the more common type there is only inversion of the mucous membrane. It is seen almost entirely in females. Bamberger has reported a case of partial prolapse into the dilated male urethra. It is due to congenital weakness of the neck of the bladder and urethra, and an exciting cause such as the strain of crying, cough, sneezing, constipation, diarrhoea or cystitis. The urethra is almost invariably dilated. The prolapse forms a red, vascular, soft, elastic, reducible tumour, about the size of a walnut and somewhat pear-shaped. It is situated below the clitoris and between the labia. It may become larger and more injected on crying and straining, and causes more or less dysuria and incontinence. The prolapse is usually of the base of the bladder, and the orifices of the ureters may be visible or



made so by gentle traction. Occasionally the anterior wall is affected. Having excluded the possibility of polypus, reduce the prolapse. It is dangerous to excise it. In Wilkinson's case the prolapse was strangulated, gangrenous, and the slough almost black. It was excised and the girl recovered. A plastic operation may narrow the neck of the urethra.

**Cystitis** is usually due to foreign bodies, gonorrhœa or *B. coli* infection; possibly exposure to cold and injury to the perineum, and less often to other organisms. It may occur in infants during infectious disease or serious illness, probably the result of retention of urine and colon infection. Some attacks are very mild and merely cause increased frequency of micturition; the urine may remain acid. In more severe ones there is frequent painful micturition, perhaps causing rigors and screaming; pain above the pubes and in the perineum, and tenderness; fever up to 103° F., enuresis and occasionally bleeding. These cases are sometimes differentiated with difficulty from bacilluria and pyelitis.

In pure *Bacilluria* the urine is cloudy, acid, and the sediment only contains organisms, seldom round cells and bladder epithelium. There may be pollakiuria. It occurs chiefly in girl babies, due to *B. coli* infection via the urethra, renal circulation or adjoining viscera. Possibly the infection is conveyed by soiled napkins, stools or threadworms. The organism can grow in acid media, does not decompose urea or cause ammoniacal fermentation, and may set up slight or superficial catarrh. It is probably fairly common, but not diagnosed through the urine not being examined unless there are symptoms. If inflammatory reaction ensues, it sets up cystitis or pyelitis (q.v.). Enuresis is of variable severity and may be only nocturnal. *Cystitis* is of two types. In one there are pallor, debility, anorexia, restlessness and fever, without local symptoms. These are more probably cases of pyelitis. In the other, a true cystitis, there are bladder symptoms in addition, viz., increased frequency of micturition, dysuria, pain in the abdominal region, bladder tenderness on pressure, and inflammation of the meatus. The urine is acid in colon infections, and not acid if due to staphylococci or streptococci. Other organisms are sometimes the cause.

*Treatment.*—Give urotropin, methylene blue, benzoate of ammonia, or salol by mouth in order to inhibit the growth of micro-organisms. In severe cystitis lavage of the bladder may be needed. Use boric acid, 3 per cent. solution for 3 days; and then silver nitrate 1 in 1000, followed by salt solution, for 3 days. The diet must be simple and contain much fluid. Alkalies are beneficial in colon infections. Possibly vaccine may do good.

**Tuberculosis** of the Bladder is said to be extremely rare under 8 years. It is probably more common and more curable than supposed. There is increasing evidence that it **may** get well by itself. It is rare as a primary disease; more common in boys, and due to extension from the kidneys or generative organs. Perhaps some primary congestion or abrasion is



necessary to permit infection, for the disease can exist in the kidneys for years without infecting the bladder. Small greyish nodules appear in the congested mucosa and undergo caseation, setting up necrosis of the superficial layers and forming shallow ulcers which may perforate.

The classical symptoms are late ones. Early signs are frequent micturition, without increase in quantity, hæmaturia, pain and pyuria; symptoms like those of stone. Hæmaturia may be the first sign, in the form of a few drops of bright red blood at the end of micturition, occurring intermittently for a few days at a time. It may exist for months without pain or frequent micturition, usually the two earliest signs and signs which are not influenced by exercise as in calculus. Hæmaturia is sometimes profuse, large clots being passed although the lesion is trivial; more often it is slight and infrequent. Pain is burning, scalding and often paroxysmal. At first it is only present on micturition, or if the bladder is distended. It extends along the urethra, not being confined to the glans as in stone, but may be referred to the base or tip of the penis or the perineum. It is most severe if the trigone is involved. With extensive ulceration, in late stages, it becomes constant in the hypogastrium. Incontinence is more common in boys than girls, and indicates that the neck of the bladder is affected.

The general symptoms are those of cystitis with loss of weight, fever, and bacilli in the urine.

*Diagnosis.*—Stain the centrifugised urinary sediment for tubercle bacilli, taking care to exclude the smegma bacillus which is present in both sexes and is not limited to the genitals; determine the tuberculo-opsonic index, if thought advantageous; and examine with the sound and cystoscope. A sound may give a grating feel as it passes over phosphatic deposit on the ulcers. The *prognosis* is bad if there is secondary cystitis, but some get well. It is unduly gloomy, because the milder cases may remain undiagnosed.

*Treatment* is mainly general. Wash out the bladder with sublimate solution, 1 in 10,000. Silver nitrate is injurious. Suppositories of opium and belladonna relieve pain. Suprapubic cystotomy may be needed.

**Vesical Calculus.**—Stone is more common in boys than girls, and among the poor. It may be composed of oxalates, uric acid, urates, phosphates or cystin. Many stones are made of calcium oxalate and primarily renal. The symptoms are frequent micturition with pain, referred to the glans penis, and the passage of blood at the end of micturition; sometimes pruritus at the meatus, retention, sudden cessation of the flow, or incontinence. Cystitis is often set up and a variable amount of pus is found, the urine becoming alkaline. This simulates tuberculosis of the bladder. Prolapse of the rectum, ulceration of the bladder and pelvic abscess are occasional sequels. The child should be examined with a sound, cystoscope and per rectum. Very highly acid urine may give



rise to the symptoms of stone, and on exploring the bladder with a sound the surface is felt rough or even gritty. It is due to the mucous membrane being thrown into rugæ and may lead to an erroneous diagnosis, but it disappears on the free administration of water and alkalies. A ureteral calculus impacted at the entrance into the bladder may cause symptoms of vesical calculus. It can be removed by suprapubic lithotomy.

Stones of moderate size and not too hard should be removed by litholopaxy. The meatus may require enlargement. If the child is under 3 years of age, or the stone large or composed of oxalates, it must be removed by suprapubic lithotomy, an operation as safe as lateral lithotomy and not likely to be followed by the serious sequels sometimes seen after the latter operation. The urine should be sterilised previously by the administration of urotropin or salol and the bladder washed out thoroughly with an antiseptic solution.

*Tumour* of the bladder is rare and usually a sarcoma. The urine may contain pus, blood and particles of tumour. The growth can be felt per rectum. It may give rise to secondary cystitis, pyelonephritis, hydronephrosis and peritonitis. Operative results are bad.



## CHAPTER XLVI.

### DISEASES OF THE KIDNEYS.

*Malformations — Movable Kidney — Cystic Disease — Hydronephrosis — Nephritis, acute and chronic — Acute Pyelitis — Calculus — Tuberculous Kidney — New Growths.*

The kidneys are relatively larger in the newborn than the adult, weighing from 12-30 gms., and for about the first 4 years are somewhat lobulated. The medullary portion preponderates over the cortical. The hilum is on a level with the second lumbar vertebra. The right kidney is 1-2 cm. lower than the left. The index finger in the rectum easily reaches the lower pole. The state of the renal organs is ascertained by palpation, percussion, inspection of the dorsum, X-rays, and the examination of the urine. Cystoscopic examination may reveal blockage of one ureter, ureteral calculus, and blood or pus issuing from the ureter. The state of the cardiovascular system and the fundus of the eye are also of assistance.

**Malformations.**—The whole urinary system may be absent or one kidney alone wanting. Complete *Absence* of a kidney is rare, 1 in 4000 autopsies. No trace was found in a 5-year old boy who died from spinal caries. Absence is 4 times as common in males as females. The left kidney is more often absent than the right. The ureter and renal vessels are absent or represented by a fibrous cord. The other kidney is increased in size sufficiently to carry on the renal functions adequately, except in serious illness. *Atrophy* is more common and may be due to thrombosis of the renal vein. The kidney is simply small and functionless, a mass of fibrous tissue, a collection of cysts or a mere nodule of fat. Calculi seem to me to develop with undue frequency in single kidneys, and uræmia may follow obstruction of the ureter. Some cases live to old age. *Fused kidneys* are “horseshoe,” “sigmoid,” or “disc shaped.” Their frequency is given as 3 in 2594 autopsies. W. G. Nash (1908) recorded a case in a girl, 16 months old, with hydronephrosis of the left half. A *supernumerary kidney* is also rare. It is usually the result of extreme segmentation on one side and may be entirely separate. Occasionally one kidney has two pelves, but the ureters are generally fused. In Watson Cheyne’s case (1899) the third kidney was movable and was removed. In Barlet’s case (1904) two left kidneys were fused together, with separate pelves, ureters and openings into the bladder.



A *displaced kidney* is found in 24 out of 1000 autopsies (Newman, 1898). The left kidney is the one usually affected. It may lie obliquely on the sacro-iliac synchondrosis, transversely across the prominence of the sacrum or between the rectum and bladder, or fixed beside the uterus. It is generally much lobulated, and associated with abnormalities of the ureters and blood vessels, and alterations in position of the large intestine and peritoneum. Sometimes fusion is present and the fused organ is unilateral, the adrenals not being displaced. It may be mistaken for enlarged glands, fæcal concretions, undescended testis, tumours, etc. Anomalies in the vascular supply are common. Occasionally there is *no renal pelvis*, the calyces entering separate tubules which unite to form the ureter.

*The Ureters.*—The ureter may be duplicated, constricted, impervious or contain valve-like folds of mucous membrane. About 40 cases of prolapse of the blind end of the ureter into the bladder, and even through the urethra, are on record. G. F. D. and A. L. H. Smith reported (1906) a case of accessory left ureter, ballooned at the lower end or ending in a cystic swelling in the bladder wall. It protruded as a dark red, globular swelling from the urethra of a girl, aged 5 days. The accessory ureter was much dilated and opened into a dilated cyst of the upper half of the kidney, the lower half of which was normal. *Idiopathic dilatation*, i.e., no obvious obstruction, is almost always associated with dilatation of the pelvis. Both may occur without dilatation or hypertrophy of the bladder. It may be unilateral. Box (1909) found unilateral ureteritis in one case. This might cause temporary obstruction of the vesical orifice. Dilatation secondary to urethral obstruction can be excluded for the bladder is normal.

**Movable Kidney.**—Mobility of the kidney is not uncommon in children. Sutherland found it in 15 out of 40 cases of orthostatic albuminuria. It may be so mobile as to be “floating,” of congenital origin due to a mesonephron. It is usually on the right side and more common in girls. Symptoms are absent; or there may be dull, aching or dragging pain in the loin, shooting down the thigh and toward the umbilicus. The pain is relieved by recumbency, and increased by exercise or constipation. Complications arise directly or indirectly from the displacement, and kinking or twisting of the blood vessels or ureters. Acute renal congestion and intense pain, leading to collapse, are due to torsion of the vessels. Frequent micturition, tenesmus, and renal colic or suppression may ensue on kinking of the ureter and transitory hydronephrosis. Pyelitis and calculus may develop; constipation, from dragging on the colon; headache, furred tongue, vomiting and jaundice, from dragging on the duodenum. The urine is normal or merely varies in quantity. It is treated by general tonics, bandages, belts and operation. In the majority of cases it may be ignored.

**Cystic Kidneys.**—Congenitally cystic kidney, unilateral or bilateral, is due to error of development. There is no evidence of intra-uterine



inflammation or tumour formation. Probably the renal changes are the same in adults, entirely developmental and differing only in degree. The secondary fibrosis is due to the tendency of malformed organs to disease. The kidney is developed from the metanephros, which forms the glomeruli and convoluted tubules ; and from the upper end of the ureter, which gives origin to the straight and collecting tubules. If the two fail to unite, the parts arising from the metanephros become cystic. They are not retention cysts arising in the uriniferous tubules from uric acid obstruction or embryonic nephritis. More than one member of a family may be affected. The surface may be entirely composed of smooth, translucent cysts, not communicating with each other. The capsule is adherent. On section there are found smaller cysts, irregular cavities separated by fibrous tissue, very little differentiation between cortex and medulla, and no traces of calyces or papillæ. The ureter may be impervious. If the urethra is impervious, the bladder is much dilated. The liver is cystic in 20 per cent. of these cases (p. 363).

The urine resembles that of hydronephrosis. Occasionally it is acid, of high specific gravity, and contains blood. The amount of urea varies and there may be a variable quantity of albumin. The disease may be so advanced as to impede delivery. Symptoms are absent in early life or are those of a renal tumour:—bulging of the abdomen in the flanks, dilated superficial veins, and a palpable tumour (in 18 out of 62 cases, Lejars).

The prognosis is bad for gradual fibrosis ensues and destroys the little renal tissue remaining. Some of the later cysts may be of similar origin to those of chronic interstitial nephritis. Calculi may form. Death is due to convulsions or uræmia. Œdema, purpuric rash and symptoms of chronic interstitial nephritis may arise, and occasionally pyuria.

Nephrectomy is not justifiable for an apparently complete cystic kidney often contains areas of healthy and partially healthy renal tissue, not visible to the naked eye. The removal of one kidney may be followed by rapid enlargement of the opposite one, though previously small. Though small it is cystic. Unilateral enlargement may be caused by calculous obstruction. Patients have lived to over 40 years of age.

**Congenital Hydronephrosis.**—This is usually bilateral, but may be unilateral. Often no obstruction is found. The chief causes are imperforate urethra, membranous septa, valves or cysts in the urethra ; developmental defects of the ureters ; or torsion of the ureter due to floating kidney. It is not necessary for the obstruction to be absolute. A valvular urethral obstruction may interfere with micturition but not with catheterisation. In a boy, 4 years old, with unilateral hydronephrosis the pelvis of the kidney was dilated and ended in a small conical apex, separated from the end of the ureter by a minute interval. Anatomically the effects vary with the causation. In bilateral cases the ureters are dilated and tortuous. The kidneys show a variable degree of cirrhosis and dilatation.



Sometimes one kidney is cystic and the other hydronephrotic. Hare-lip, club-foot and other deformities may be present.

The condition may interfere with labour. The child may be stillborn, or live for weeks to years. Most of them die in early infancy. Out of 10 cases 6 died under 6 months and only 2 lived 2 years (Holt). It gives rise to a globular, more or less cystic swelling extending into the loin; perhaps so large as to pass the middle line and yield a thrill. It is often not diagnosed during life for genito-urinary symptoms are usually absent. The urine from some cases has contained little or no urea. If unilateral and increasing in size, sufficiently to cause symptoms, it should be removed.

**Acquired Hydronephrosis** is usually the result of calculous or other obstruction, external pressure on the ureter and, rarely, ureteral stricture such as may follow septic infection and surgical kidney. If the obstruction is variable in degree it is apt to give rise to attacks of pain in the abdomen and renal region; tenderness, rigidity and fulness; and scanty micturition. Such an attack may end with the passage of a large quantity of urine of low specific gravity. Hydronephrosis must be diagnosed from simple cyst, cystic disease, tumour, pyonephrosis and tuberculous kidney.

**Nephritis.**—The urine should be examined in fever and acute disease for nephritis is a common complication and albuminuria still more frequent. Albuminuria, even with œdema, is not proof of nephritis. Both these signs, and possibly hæmaturia also, may be present apart from actual inflammation of the kidney. On the other hand nephritis may be found post mortem although no albuminuria was present during life. In nephritis the diagnosis is based on the urinary analysis and associated symptoms, as in adults: viz., blood, albumin, casts, alterations in the quantity, colour and specific gravity; cardio-vascular changes, retinal changes, œdema, and complications affecting the serous membranes. It is almost impossible to make an exact diagnosis of the special variety of renal disease by urinalysis. The want of correspondence between clinical symptoms and renal changes is a frequent and striking characteristic. The causes of albuminuria without nephritis have been already mentioned and discussed (pp. 551-556).

*Infantile œdema* is by no means always due to nephritis, e.g., œdema neonatorum (p. 146), which develops within a few days of birth and is analogous to phlegmasia alba dolens in some instances, due to thrombosis of the vena cava inferior. *Idiopathic or essential œdema* (L. F. Meyer, 1905) is a type in which neither albumin nor casts are found in the urine, and the kidneys in fatal cases appear unaffected. It is increased by adding common salt, gm. 1-2 daily, or sodium phosphate to the diet. Meyer ascribes it to a functional derangement of the renal epithelium, whereby the salts are imperfectly excreted and, accumulating in the tissues, cause retention of water (Hutinel's theory). Some cases can be cured by increasing the percentage of protein in the food. To a similar condition S. West gives



the name of *pseudo-renal dropsy*. It occurs in infants and young children with marasmus or malnutrition, anæmia, and often prolonged diarrhœa. It may become extreme in a few days and even cause serous effusions. No nephritis is found after death. Digitalis is useful. An *acute general œdema* is occasionally seen in infants and children, for which no cause can be ascertained and one is reduced to a diagnosis of some kind of toxæmia affecting the blood and possibly the renal epithelium. I have noted in a few cases of this type that the secretion of urine was scanty at the onset and that a trace of albumin and a few red cells were present; but the child recovered in a few days. Perhaps these are instances of *renal urticaria*. Generally speaking we must regard œdema in the above instances as dependent on altered states of the blood or on malnutrition of the renal cells, causing a renal inadequacy. These remarks must be read in conjunction with those on œdema as a symptom (p. 457). Œdema, albumin and hyaline casts can occur in the newborn without nephritis. The amount of œdema depends on the scantiness of the urine. The prognosis is that of the cachexia.

**Nephritis in Infancy.**—It may occur in the newborn and be even intra-uterine in origin. Ashby reported the case of an infant who developed œdema of the face on the second day of life, followed by general œdema, diarrhœa, vomiting, oliguria, and death from convulsions at 4 weeks of age. Nephritis of the type of scarlatinal nephritis was found after death. G. Carpenter and Henoch have recorded cases in infants 5 weeks old, and Jacobi mentions 5 cases seen within the first 5 weeks of life, for which no cause could be discovered except prolonged asphyxia at birth. One of Carpenter's patients was syphilitic. Nephritis in infants may be of gastrointestinal origin or due to other infections and intoxications. The contracted kidney is a curiosity at this age and nephritis due to congenital syphilis very rare.

*Gastro-intestinal nephritis* may occur in any acute attack of infective diarrhœa, possibly in toxæmic diarrhœa. It is degenerative, if of toxic origin, or hæmorrhagic in type. Albumin, casts, renal epithelium and blood cells may be found in the urine. The chief symptoms are debility, sighing, vomiting, diarrhœa, tense fontanelle, œdema and convulsions; or anuria, œdema, rapid pulse, high temperature and slight dyspnœa, which may end in vomiting, restlessness, screaming, convulsions and drowsiness or coma. Many of the fatal nervous symptoms and the hydrocephaloid state may be of uræmic origin. The urine is suppressed or scanty. The kidneys recover in toxic cases as the primary disease gets well. In bacterial infections the course is more prolonged and the disease may become chronic. Hæmaturia and infarcts may be due to thrombosis of veins; and death may ensue from uræmia. Acute nephritis in infants from other causes presents similar symptoms. It may simulate encephalitis or meningitis. The prognosis is grave. Out of 23 cases under 2 years of age 11 died (Holt).



The kidneys show fatty degeneration of the epithelium of the convoluted tubules and swelling of that of the straight tubules; areas of infiltration of round cells and erythrocytes in the renal cortex; and organisms in the vessels and exudate. In 5 of Holt's fatal cases the nephritis was interstitial, and in 5 parenchymatous.

*Varieties of Nephritis.*—All types are seen in early life, running much the same course as in adults. Some are primary, others secondary and occurring in the course of another disease. The inflammation is acute, subacute or chronic. The anatomical changes are parenchymatous (glomerular or tubular), interstitial, or diffuse and mixed. Thus acute nephritis may be of any of the above pathological types, and primary or secondary. Many cases are really chronic ones with sudden exacerbations. It is a bilateral affection with invariably a certain amount of interstitial change. It terminates in recovery, chronic nephritis or death. The diffuse or mixed type produces the diffuse or "large white" kidney; if there is much interstitial change, it ends in the "contracted white" kidney. The latter variety may possibly depend on a tubular inflammation supervening on a primary interstitial nephritis.

**Acute Nephritis.**—This is glomerular or tubular, according to the main distribution of the inflammation. The type is only distinguishable after death. The glomerular variety is the one so often seen in scarlatina and other infections.

*Etiology.*—Primary and secondary nephritis are quite frequent in children, especially at the age of 3-9 years. The sex-incidence is 3 males to 2 females. They are most common in temperate climates. Primary nephritis may be due to mechanical injury during or after birth, renal infarcts and calculi; circulatory disturbances such as chills and cold baths, extreme heat, too hot baths, asphyxia neonatorum, embolism, infarcts, and hæmorrhagic effusions in purpura and scurvy; toxic causes, e.g., turpentine, cantharides, mercury, storax, balsam of Peru; toxins derived from the throat, intestines, etc. A history of sore-throat is common.

Secondary nephritis occurs in the course of any specific infection, especially scarlatina, measles, influenza, variola, diphtheria and, less often, typhoid, varicella, mumps, glandular fever, pneumonia, pertussis, acute rheumatism, erysipelas, general sepsis, and syphilis. Diphtheria is more likely to give rise to albuminuria from toxic degeneration of the tubular epithelium or cloudy swelling, similar to what happens in high fever. The differentiation between the albuminuria of fever or toxæmia from that of nephritis is not always easy.

**Scarlatinal Nephritis** is the type of acute nephritis in early life. It must be distinguished from the febrile albuminuria with casts in scarlet fever, and from the septic nephritis which occurs in septic scarlatina and other septic states. Its frequency varies in different epidemics. Ashby found it in 6-7 per cent. of over 3000 cases. Caiger states that it was



present in 2.8 per cent. of over 67,000 cases ; but he found it in 77 out of 2148 cases in hospital. A family predisposition has been noted.

It begins at the end of the second or during the third week, that is at a time when the scarlet fever is over. The initial albuminuria may have been absent for some days. About half the fatal cases begin in the first week. Sometimes the onset is delayed until the fourth week. The onset may be preceded by a fall in specific gravity of the urine ; an increase in quantity and frequency of micturition ; a slight rise of temperature and excess of urates ; or commencing œdema and increased arterial tension. It may be ushered in with fever ; hæmaturia, or albuminuria at first and hæmaturia in 2-3 days ; or with vomiting, fever, anorexia, pallor and puffy eyelids. The vomiting is a bad sign, if it is persistent. The severity of the onset varies from scarcely any disturbance to a severe illness with nausea, vomiting, anorexia, headache, restless sleep, rapid pulse, a temperature of 104-106° F., rigor and sweating. Sometimes the pulse is infrequent though the temperature is high. Pallor, swelling of the face and œdema over the tibiæ are early signs.

The *urine* is turbid, high-coloured, of high specific gravity, 1025-1040, and later falls to 1015. At first it may be clear though containing albumin, casts, red and white cells, and renal epithelium. Casts may be present without albumin. The colour depends on the quantity of urine and the amount of blood. It may be solid with albumin on boiling and the coagulum a dirty brown. The deposit contains many red cells, microcytes, hyaline casts of all sizes, granular, epithelial and blood casts, renal epithelium, leucocytes and fatty cells.

*Morbid Anatomy.*—Sometimes the kidneys are large, red and vascular. Usually they are pale, mottled and streaked with dark red. The capsule peels off easily and leaves a smooth surface. The cortex is wide and ill-defined, and the arteries are not thickened. The whole organ is involved, some parts more than others, in an inflammatory engorgement or hyperæmia. It is an acute interstitial nephritis, hæmorrhagic in character, which may extend only to the glomeruli (glomerulo-nephritis or hæmorrhagic glomerulitis) or be uniformly distributed. The glomeruli are swollen and crammed with red cells, with some small celled exudation between the capillaries. Similar exudation is found between the tubules and the vessels are engorged. Exudation takes place into the tubules, which become blocked with coagulated blood and fibrin, causing oliguria. The epithelium of the glomeruli is cloudy, swollen and finely granular. Later, the cells undergo fatty degeneration, and granular and fatty epithelial casts are found in the urine. Sometimes the streptococcus pyogenes can be cultivated from the kidney. If recovery is imperfect, fibroid interstitial changes occur about the glomeruli and throughout the kidney.

In primary nephritis the main change is round and in the tubules, the glomeruli not being affected more than the other parts. The changes



are primarily vascular and are followed by degeneration of the renal epithelium, regeneration, and the formation of new fibrous tissue.

*Course and Complications.*—The attack may be mild, severe or very severe. In mild cases there is a little fever, with slightly smoky urine, and the attack clears up in a week or two. In a more severe one there is scanty urine, much albumin and œdema. The attack ends rather abruptly in 1-3 weeks, but slight albuminuria may reappear during convalescence, when the patient gets up, or may persist for weeks and months; usually running a fairly definite course and ending in 1-3 months. In the worst cases the temperature is continuously raised, the urine very scanty, the amount of albumin up to one-third or more, and the œdema is extreme and may affect the mucous membranes, e.g., the glottis. Dropsical effusions into all the serous cavities and uræmia may develop, with epistaxis, twitchings, convulsions and coma. These attacks are associated with complications, such as pericarditis, myocarditis, cardiac dilatation, peritonitis, pleurisy, pneumonia and even gangrene of the lung. All the tissues of the body are water-logged. The dropsy is due to impaired nutrition of the arterioles or to cardiac failure. Death is caused by suppression of urine, uræmia or complications.

The heart is often involved quite early; generally the left ventricle is dilated and there is a small frequent pulse, precordial pain and dyspnœa. The dyspnœa may come on quite suddenly, when due to cardiac failure. It is called uræmic asthma or cardiac dyspnœa. It may depend on the uræmia or on pulmonary œdema, occurring in dropsical cases or in uræmia without dropsy. Progressive anæmia is often present.

*Diagnosis.*—In one type of case there is marked dropsy; in another it is absent. In the latter variety the nephritis is apt to be overlooked and the albuminuria regarded as simple. Microscopical examination of the urine is essential. Scanty blood-stained urine is due to acute nephritis, extreme passive congestion or infarction. In cases of *infarction* the onset is sudden, with perhaps copious albuminuria and no blood, no dropsy, evidence of a source of embolism or thrombosis, and sudden subsidence. Œdema may be secondary to heart or lung complications. It can occur in oliguria without albuminuria, although there is scarlatinal nephritis, as shown by peeling and subsequent urinary changes. Œdema is most frequent in glomerular nephritis and may be associated with effusion into serous cavities, uræmia and convulsions, and yet end in recovery. A past history of scarlatina, a history of other cases in the house or neighbourhood, and peeling are important aids in the diagnosis of scarlatinal nephritis from other varieties.

*Prognosis.*—Few cases are fatal and uræmia is rare. Most patients recover in 1-3 weeks, if there is no dropsy or uræmia. The urine becomes clearer and more plentiful, and albumin disappears; sediment on standing persists longer. In worse cases the dropsy spreads and becomes general;



and the kidneys are enlarged and painful on pressure. Even serious cases of dropsy and uræmia recover. The outlook depends on the general condition. It is bad if the heart is involved; and the gravity is increased by high fever, anuria, alimentary troubles, and tachycardia or bradycardia. Inflammation of the lungs or pleura and uræmia are the chief causes of death. There is considerable liability to exacerbations and relapses. Some cases become chronic, but it is rare for an acute attack to pass at once into the chronic variety. The prognosis is influenced by the age of the child and the period of the fever at which it begins. If the fever has subsided for some time before the onset, the outlook is favourable. Death is rare under 10 years of age. There is hope for cure as long as red cells are still present. The night urine first becomes free from albumin.

*Septic Nephritis* may be primary, without any evident source of infection, but is generally due to the septic complications of scarlatina. The inflammation is local rather than general; great exudation of leucocytes from the vessels, coagula crowded with cocci in the tubules, much epithelial degeneration, minute hæmorrhages and small points of suppuration at the bases of the pyramids. Albuminuria without hæmaturia is found; sometimes casts and pus. There is no œdema and rarely uræmia. Vomiting, occasionally diarrhœa, anæmia, bronchial catarrh and irregular fever are often present. A septic nephritis, due to ascending urinary infections, is indistinguishable from pyelonephritis. Sometimes during the first month of scarlatina a virulent hæmorrhagic nephritis occurs. It runs a rapid course and is very fatal. The urine may be normal until within a day or two of death.

*Influenzal Nephritis* is sometimes of the ordinary glomerular type, more often hæmorrhagic. It is usually an early complication but may appear at any stage. Œdema, not often marked, is present in about half the cases. The attack is over within 3 weeks and the prognosis good. It may become chronic. The toxæmia of severe influenza may cause albuminuria, or hæmaturia only.

*Diphtheritic Nephritis* is a toxic nephritis with slight tendency to œdema and uræmia, and few general symptoms. The convoluted tubules and descending limbs of the loops of Henle, and next the collecting tubules, are chiefly affected. The change is a fatty epithelial one, purely parenchymatous. Hæmorrhages are rare. The urine is of normal colour, high specific gravity, and contains hyaline and epithelial casts, leucocytes, fat cells, and few red cells. It is really a toxic degeneration which may be associated with a mild degree of hæmorrhagic nephritis. Recovery takes place in 1-2 weeks.

**Primary Nephritis** is insidious in its onset, but may be acute and ushered in by vomiting, headache, general malaise, a rise of 2-3° F. in temperature for a few days, and urinary changes. Puffiness of the eyes, swelling of the face and œdema are often the first signs. The symptoms are



less severe than in adults, headache less common and backache rare. Blood pressure rises from 60-120 mm. of mercury. Dropsy may be slight. Some attacks are very short. Others run a long subacute course, and the prognosis is not as good as in the scarlatinal type. An average duration is 1-3 months. Convalescence is often prolonged and albumin may appear from time to time for 2 years. Few chronic cases are seen and recurrence is rare. Death may ensue from high fever, complications, uræmia, convulsions and coma. The chief complications are serous effusions and inflammations, pneumonia, toxic gastro-enteritis, and rarely œdema of the glottis and lungs, amaurosis, œdema of the discs and optic neuritis. Many of these attacks are no doubt of infectious origin, although the course of infection is not found, and are hæmorrhagic in type.

**Chronic Parenchymatous Nephritis** is the sequel of an acute or subacute attack of primary or secondary acute nephritis. It affects 3 boys to 2 girls. Possibly there is an hereditary tendency to renal weakness, for Heubner has met with it in several members of the same family and I know of a family in which 4 children were affected after scarlatina. The common age is 6-14 years, but it may occur earlier and even in infancy. The usual variety is the large white kidney. Some cases are insidious in onset, with nothing to suggest an acute attack. Probably some of these result from mild unsuspected scarlatina. The *symptoms* and course are the same as in adults. The urine is scanty, highly albuminous, and contains hyaline, granular, fatty and epithelial casts. The main symptoms are pallor, puffiness, extensive œdema, vomiting and general malaise. There is much liability to bronchitis and pulmonary affections, and to serous inflammations and exudations, occasionally retinitis. The urine may clear up on milk diet and rest in bed, but almost invariably relapses on getting up.

No case is hopeless. A few are cured although they have gone on for years. Recovery is rare if albuminuria has persisted for one year. The disease is essentially chronic and may last for 20 years. These prolonged cases, in which the urine persistently contains small quantities of albumin and hyaline casts, are somewhat different. The quantity, colour, reaction and specific gravity are normal. There are no symptoms and no ill effects beyond slight pallor and loss of tone. Probably a limited area of renal tissue only is damaged. It is impossible to give an accurate prognosis as the amount of kidney permanently damaged cannot be estimated. The quantity of albumin and fatty casts affords a fair guide to the severity of the case. As long as the child does not become distinctly anæmic the outlook is not bad.

If the urine increases in quantity and diminishes in specific gravity it is probable that interstitial changes are taking place, producing the contracted white kidney. Cardiac hypertrophy, raised blood pressure and thickening of the arteries, possibly other signs, will then be found. Occasionally a *chronic hæmorrhagic* variety is seen in which there are



acute exacerbations with hæmaturia and scanty urine, and no œdema. These are less serious.

**Chronic Interstitial Nephritis** or "*granular kidney*" is seen at times in the young. The sex-incidence is 2 males to 3 females. Nettleship (1904) collected 80 cases under 21 years of age. J. E. H. Sawyer (1907) collected 55; males 22, females 33. Of Sawyer's cases 23 were under 10 years, 21 more under 15, and the remainder under 21 years of age.

Its *etiology* is doubtful. It has been regarded as hereditary (4 generations, Dickinson), congenital, due to hereditary syphilis, or a sequel of infective disease, notably scarlatina and less often measles and varicella. In some instances there has been a history of alcoholism and a few have been due to renal calculus. It may be unilateral or much more marked on one side than the other. Probably most cases are primary and of insidious onset; and others are secondary to mild unrecognised nephritis in infancy.

The *symptoms* and *physical signs* are the same as in adults, but, not being suspected, are often overlooked. The child comes under observation for wasting, gastro-intestinal catarrh or cough. Or thirst, polyuria and enuresis are the first symptoms, though not always prominent. A few are ushered in by convulsions and coma. The child is listless, small, stunted, wasted and anæmic; the cheeks may show a dusky flush. The skin is harsh and dry, devoid of sweat, coarse, inelastic and pigmented, of a dirty, brownish yellow or brown colour, suggestive of Addison's disease. The pigmentation, if present, is chiefly on the abdomen, flanks, axillæ and groins. The heart is hypertrophied, the aortic second sound accentuated, and the arteries thickened. Dropsy is almost always absent; a little may result from cardiac failure. The mental state is precocious, sometimes perverse.

The cerebral symptoms are headache, vomiting, vertigo, restlessness, mental disturbance, tremors, tetany, convulsions and those of cerebral hæmorrhage. Amaurosis, diplopia, retinitis and retinal hæmorrhage may be present. The gastro-intestinal signs are thirst, vomiting, diarrhœa, obstinate constipation, and abdominal pain; pulmonary ones are bronchitis, dyspnœa, œdema and asthma; cardiac ones, precordial pain and dyspnœa. Many of these symptoms may be absent. Severe bleeding after tooth extraction has been noted. Epistaxis may occur early from toxæmia. The morning headache is due to the accumulation of toxins during the night, or altered cerebral circulation, sudden tension after relaxation during sleep. The vomiting may be the result of similar causes or purely gastric.

The urine is profuse, 3-5 pints, pale, specific gravity 1004-1020, and contains a small, variable amount of albumin, and a few hyaline and granular casts. Hæmaturia may result from an exacerbation and even severe bleeding may occur.

Cases are mistaken for enuresis, diabetes insipidus, functional albuminuria, the uric acid diathesis, and simple malnutrition. A mild attack



of hæmaturia may be diagnosed as acute nephritis but there is no œdema, and the amount of blood and albumin fluctuate from day to day.

Death results from uræmia; occasionally from cerebral hæmorrhage or some complication. The duration of life depends on the duration of the disease previous to diagnosis, and this cannot be estimated accurately. It varies with the degree of cardio-vascular changes and the possibility of maintaining cardiac hypertrophy.

**Eye Symptoms in Nephritis.**—In acute nephritis eye changes are uncommon. Optic neuritis is the most frequent. Uræmic blindness is probably central and toxic. Retinal hæmorrhage may occur. Albuminuric optic neuritis is diagnosed with difficulty from other varieties unless there are retinal changes, such as hæmorrhages, old or recent, patchy atrophy of the retina, or acute inflammation and effusion of lymph.

Albuminuric retinitis is quite rare in children and seldom seen in acute nephritis. It is a true inflammation and liable to occur in the chronic parenchymatous type (10-15 per cent.). The retina is opaque and hazy; the optic discs swollen; and there are opaque, white, non-glistening patches of exudation obliterating or obscuring the vessels, and few hæmorrhages. It may get well, but the prognosis is probably as bad as in adults.

The degenerative type of retinal change is seen in cases of granular kidney (5-10 per cent.). It is due to arterial disease and hæmorrhage. The arteries are thick and look like silver wire. The hæmorrhages are distributed along the vessels or as flame-shaped patches between the nerve fibre layers. Brilliant glistening white patches of choroido-retinal atrophy are scattered about the macula and radiating from it. White patches in the retina may be due to exudation of lymph, old hæmorrhages, or atrophy of the retina and choroid. Optic neuritis is absent or slight. The retina may become detached. Vision may remain good, if the macula is little or not at all involved. The extent of vision is variable and complete blindness rare. The prognosis is grave. Hæmorrhage indicates advanced disease and a short life. Death may follow in a few months or not for 3 years, but life is rarely prolonged beyond a year.

**Uræmia** is latent, acute or chronic. It is due to the accumulation of products of metabolism, which should be excreted by the kidneys, or to the failure of an internal renal secretion, or to cytotoxins formed from degenerated or diseased renal cells. It is noteworthy that symptoms are absent in hysterical anuria, lasting for days or weeks, and in anuria from calculous obstruction unless there are secondary renal changes. Ammonia, urea, creatinin potassium, chlorides and acids have all been excluded as causes. Urea is excreted by the intestines when the kidneys fail. In uræmic diarrhœa the fæces contain much ammonia; and in renal disease, an excess of nitrogen over that in the food. Ammonia is also found in the stomach.

Acute uræmia is most frequent in scarlatinal nephritis, but may occur in granular kidney. The chronic form occurs with the granular and the



large white kidney. The latent type is that of calculous anuria, obstructive suppression from blockage of the ureter, reflex arrest of the functions of the kidneys, or destruction of the cortex in cystic disease and hydro-nephrosis. It is the same as that produced experimentally in the laboratory. Penrose (1904) reported a case of uræmic fits in a boy, 1 month old, causing tonic spasm of the fingers and hands, fixation of the eyes, occasional rigidity of the head and neck, cyanosis, slow and laboured breathing, but no general convulsions. Death occurred next day. The foreskin showed a pin-point phimosis. The ureters were enormously dilated and filled with greenish purulent urine; the bladder a little dilated and contained similar urine; and there was advanced pyelitis, and multiple abscesses in the renal cortex.

*Latent Uræmia.*—The urine may not be notably reduced in quantity; perhaps it cannot be estimated, through being passed in bed. The temperature is subnormal, pupils small, vomiting occasional, and the child conscious, sometimes irritable and restless. Dyspnœa, convulsions and coma are absent. Uræmic dyspnœa may occur in calculous anuria with pyelo-nephritis. Toward the end there may be slight muscular twitches and some drowsiness. The duration is from 5-9 days.

*Acute Uræmia* exhibits nervous symptoms, with or without those of alimentary disturbance. Convulsions and coma may come on suddenly, simulating epilepsy. The pupils are contracted. More commonly the signs are anorexia, ammoniacal breath, thickly furred tongue, vomiting, headache, dyspnœa, restlessness, anxiety and nervousness, dilated pupils, frequent pulse, subnormal temperature, muscular twitches and scanty urine. These symptoms are followed by drowsiness and convulsions. Between the fits the child may be comatose, or violently excited and restless. Gradually the drowsiness increases; the deep, jerky respirations assume the Cheyne-Stokes type; the pulse becomes more frequent; and twitchings, convulsions and coma end the scene, or death may occur suddenly. Sometimes there is great weakness, epigastric discomfort and inability to swallow. Uræmic blindness may come on quite suddenly, without any fundus change.

The features of uræmia vary according to the predominance of cerebral or motor symptoms. The respiratory peculiarities are due to the action of the poison on the respiratory centre, for even in severe dyspnœa the lungs may show nothing abnormal. Vomiting is partly cerebral, partly gastric.

Acute uræmia can occur although large quantities of urine and urea are evacuated, and in the absence of albuminuria. The prognosis depends on the stage of the renal disease and the absence of complications. It is fairly good in scarlatinal and other forms of acute nephritis, except when grafted on chronic renal mischief. After a few convulsions recovery may ensue. The fits may be accompanied or followed by amblyopia, amaurosis, aphasia, local palsies, or various psychoses.



*Chronic Uræmia* is indicated by some of the following signs :—languor and torpidity, restlessness, sleeplessness, nervousness, vertigo, headache ; dirty tongue, attacks of vomiting, perhaps diarrhœa, hiccough ; dyspnœa or uræmic asthma ; slight twitchings, first noticed in the muscles of the thumb ; and, less often, itching of the skin, drooping of the eyelid or some other paresis, noisy or muttering delirium, delusions, gradual unconsciousness, coma and even convulsions. The duration is about 10 days. It is rarely recovered from.

**Treatment of Renal Disease.**--Reduce the work the kidneys have to do, by limiting the diet, especially protein foods ; and replace their activities by increased action of the skin and bowels. In acute nephritis bed is essential, flannels and blankets for clothing, and the horizontal posture to limit the strain on the heart. A diet of sugar (5-7 oz. for adults) and water only is recommended by v. Noorden. Generally it is permissible to give hot tea, mineral waters, milk, fruit, and carbohydrates, or diluted milk alone in the acute stage. Prohibit meat extracts. Warm baths, and counter-irritation in the lumbar region by mustard leaves, poultices or dry cupping, will relieve the renal congestion. Keep the bowels freely open, without excessive purgation, by mag. sulph., sod. sulph., or pulv. jalapæ co. Do not give mercurials. Internally, let the child have plenty of water, imperial drink, and drugs such as effervescing citrate of potash, acetate of ammonia, and the tartrate, phosphate and benzoate of soda. The addition of sod. bicarb. and pot. citrat.,  $\overline{aa}$  grs. 2-5, to each milk feed is beneficial.

When the acute stage has subsided and there is no longer blood in the urine, give more active diuretics, viz., tincture and liquid extract of jaborandi, digitalis, strophanthus and sal volatile, and caffeine. Fish and eggs can be added to the diet, and subsequently chicken, calves head and other proteins, if the albumin does not increase. A much restricted diet, too long continued, impairs the general health and retards recovery.

Subsequently, and in all cases of large white kidney, rely on iron. It may be tried tentatively, in conjunction with digitalis, in prolonged cases before the hæmaturia has entirely ceased, provided it is at once omitted if the blood increases. The best results are obtained with tr. ferri perchlor. In one case it was given for over 3 years and the patient recovered. The chief drawbacks to its use are the discoloration of the teeth and the liability to upset the digestion. Iron must be used with caution, if there is any cardio-vascular change, and given in milder forms.

For *œdema* give hot air or vapour baths, diuretics and purgatives. Care must be taken to avoid scalding. The fluid intake and the amount of salt should be reduced, or a salt-free diet tried. After the first year of life, diuretin grs. 2-10, q.i.d., is a useful diuretic, if the œdema is due to cardiac failure. It apparently does not injure the renal epithelium. In infants it may cause gastro-intestinal derangement. Other diuretics are available. Pilocarpin and jaborandi induce profuse sweating and are



rarely advisable. If there is excessive effusion, it may be necessary to tap the legs with Southey's trocars, taking the utmost care to prevent secondary sepsis. As the œdema diminishes the kidneys improve.

*Uræmia*, if acute, must be actively treated by hydragogue cathartics, vapour baths and pilocarpin, provided it is associated with œdema. Otherwise the treatment is apt to make the patient worse by concentrating the blood and the poison therein. For twitchings and convulsions give chloral per rectum, bromides, chloroform inhalations, or minute doses of morphia. Venesection may induce the cessation of the fits and abundant urination. It is indicated in early stages showing brain irritation, whether the child is robust or weakly. It does most good in first attacks during scarlatinal nephritis. Saline injections are also useful. Decapsulation has been used in acute and subacute cases which do not yield to treatment, especially in commencing uræmia. It is applied to both kidneys. It reduces tension and drains the œdematous tissues. Possibly the benefit is due to the latter result. A new capsule forms in 3-4 months. It is extremely difficult to estimate the value of treatment in acute nephritis for many cases are mild and recover under the simplest measures. I am strongly opposed to the use of an icebag locally and even dry cupping is unnecessary, unless there is anuria.

*Granular Kidney*.—Rely on general and symptomatic treatment:—hot or Turkish baths, suitable exercise, avoidance of chill, warm clothing, and the moderate use of purgatives, diuretics and diaphoretics; a limited nutritious diet, with little protein food; strophanthus and ammonia, digitalis and tonics for cardiac failure; pot. iod., nitrites or trinitrin for high tension, headaches, insomnia and irritability. These patients must live in a warm equable climate.

**Acute Pyelitis or Pyelonephritis** is primary in infants and secondary at all ages. The rarity of the disease in infants varies inversely as the frequency of urinalysis and the difficulty of obtaining the urine. Pyelitis, pyelonephritis and pyonephrosis are progressive stages. Pyelitis may be limited to the pelvis of the kidney, or may be secondary to and associated with cystitis.

*Etiology*.—The common causes are calculous irritation; drugs such as turpentine, cantharides, carbolic and salicylic acid; *B. coli* infection of retained urine; gonococcal and other infections extending upward; pyæmia and acute infectious diseases. Infantile cases are generally seen in girl babies, usually bottle-fed, under 2 years of age. They are due to microbial infection per urethram or from the alimentary tract, secondary to an acute gastro-enteric infection, via the blood or lymphatics. Often there is a history of constipation. Excoriation or fissure of the anus was found by John Thomson in 5, and some soreness of the vulva in 2, out of 8 cases.

*Symptoms*.—The onset is sudden with fever, shivering, blueness of the face and cold extremities; or even rigors, restlessness, and spasmodic



colicky pains. Sometimes there is headache, drowsiness, and delirium; occasionally vomiting and diarrhoea. The fever is high, perhaps 104-106° F., and runs a course very suggestive of typhoid fever, or more like pneumonia, and at times with intervals of apyrexia. Its duration is variable; it may last for 2-4 weeks. Rigors are rather characteristic, if they occur, for they are rare in infancy. The pain is variable in character, severity and distribution. It is usually in the loins or subcostal, and often unilateral. It may induce screaming attacks, frequent micturition and defæcation. Tenderness may be found over one kidney, generally the right. There may be anorexia or an unimpaired appetite, regular action of the bowels, and no marked wasting unless the attack is prolonged. The child becomes very pale. Anorexia, irritability, extreme pallor and occasional fever may be the only symptoms. Rarely there are convulsions, opisthotonos, coma, high fever, urinary intoxication, septic infection and a fatal issue.

The urine is increased in quantity, slightly turbid or opalescent, not clearing on heating, and acid. It contains a variable amount of pus, rarely much, and a little albumin or nucleo-protein which is due to the pus, some epithelial cells from the pelvis and ureters, distorted white and crenated red cells, and crowds of motile bacilli which prove to be *B. coli* in 90 per cent. The urine is turbid, even after filtration through filter paper, for the organisms pass through though the pus cells are retained. There may be partial suppression at the onset. Some cases show crystals of uric acid or oxalates. There are no casts.

The attack lasts for 2-6 weeks; pyuria perhaps for months. Sweating is marked in some cases and splenic hyperplasia is not uncommon. It is rarely fatal. Convalescence is tedious.

*Diagnosis.*—The illness closely resembles typhoid fever clinically. Possibly some cases are really due to paratyphoid or paracolon infection. Cystitis is indicated by frequent and painful micturition. In ordinary pyelonephritis the urine is acid and contains much pus, albumin and many organisms. In nephritis there are casts, renal cells, much albumin, and scanty urine. Pyuria is not infrequent in convalescence from pneumonia, typhoid fever, measles and diphtheria; and in delicate and anæmic children. In small amounts it does not mean pyelitis. It is necessary to exclude pus from the urethra, bladder, vulva, vagina, or an abscess opening into the urinary tract; renal calculus and tuberculosis. The acute onset suggests influenza and the illness is alarming and undiagnosed unless the urine is examined.

*Pyelonephritis in children* may be of three types:—(1) Acute; with rigors, fever, lumbar pain, abdominal pains and sweating. The urine is unaffected for 2-3 days and then shows pus and organisms. Sometimes there is retention, occasionally blood. (2) Subacute; with unexplained fever, anorexia, malaise, anæmia and wasting. (3) Chronic and relapsing;—recurrent attacks with fever, headache and vomiting, malaise, painful micturition, and perhaps lumbar pain, suggestive of stone.



*Treatment.*—Potassium citrate, grs. 24-48 daily, is useful for the infantile disease. It renders the urine alkaline and thus hinders the growth of the organisms, for *B. coli* will not grow in an alkaline medium. Unfortunately it causes nausea, depression, flabbiness and low temperature. Urotropin, excreted as formalin in the urine, and its allies are chiefly of value in the bacteriuria of typhoid fever and in pyelonephritis, and much less efficacious in infantile pyelitis. It must be given in a large quantity of water, e.g., grs. 5-10 in water oz. 4. Methylene blue, creosote and salol, excreted as a phenyl compound, are sometimes beneficial.

**Renal Calculus.**—Calculous concretions may be found at any age in the kidney, ureter, bladder or impacted in the urethra. Uric acid concretions in the kidneys in the first few days of life have been already described (p. 548). At a later age an excess of uric acid crystals in the urine is due to a gouty heredity, cold, unsuitable diet, digestive and hepatic disturbance, and scurvy. It is aided by deficient cutaneous excretion, the result of dirt or unsuitable clothing, hot days and chilly nights, and deficient supply of fluids. Most renal calculi are composed of urates or uric acid; a few of oxalate of lime, and, still more rarely, cystin. They are comparatively rare; vary in frequency in different localities; more common in boys than girls, and at 2-6 years old; and may be present in large numbers.

The passage of gravel (uric acid) or oxalates may cause symptoms of stone; viz., pain and tenderness in the lumbar region, dysuria, pain referred to the end of the penis, and attacks of colic which are generally mistaken for intestinal colic; sometimes irritable bladder and enuresis; hæmaturia, albuminuria and crystals. The urine may be very pale, of low specific gravity, and contain very little urea. The pain of lithiasis and oxaluria is usually bilateral, that of stone is unilateral.

The passage of crystals may give rise to renal colic in quite young infants; with paroxysms of pain, vomiting, drawing up the legs, tenderness in one or both lumbar regions, anuria or retention, or the frequent passage of urine containing crystals, mucus and blood. Shrieking attacks may be the only symptom. Sometimes there is violent spasmodic pain, with dysuria, priapism and even convulsions; the attack ending suddenly with polyuria.

*Symptoms* of stone may be absent if it is in the substance of the kidney, and even when the pelvis and calyces are full of stones. The pain depends on their position and mobility. The pelvis and its branches are very sensitive, the renal substance insensitive. It is unilateral, variable in character, increased by exercise, and may be renal colic. The pain comes on suddenly in the lumbar region or hypochondrium, and runs down the ureter to groin, bladder and penis. Retraction of the testis is more common in boys than men. The colic is due to temporary obstruction at the mouth of the ureter and backward renal distension, clots of blood in the pelvis and passing down the ureter, or the passage of a stone.



Tenderness is found over the kidney, and perhaps blood in the urine after the manipulation. Hæmaturia is often absent; sometimes the only symptom and may be profuse; is excited or increased by exercise, and is apt to follow renal colic. Micturition is often frequent, sudden and uncontrollable, but by no means always so. It may be reflex in origin, or due to the blood or pus. Reflex distension of the bladder may occur; so, too, retention or anuria. Many of the symptoms are those of pyelitis or pyelonephritis. There is rarely fever. Gravel may be found in the urine.

*Diagnosis.*—Examination with X-rays generally enables the stone, if present, to be localised in the kidney or ureter. It is much more often in the ureter than formerly supposed. Oxalate stones give the best, and uric acid ones the worst shadows. It has to be diagnosed from simple lithiasis and oxaluria, intestinal and appendicular colic, caries of the lumbar spine, movable and tuberculous kidney. The chief *sequels* are hydro-nephrosis, pyonephrosis and latent uræmia. Renal colic is less severe than in adults.

*Treatment* is more medical than surgical. Nephro-lithotomy is rarely needed. Give alkaline waters, e.g., Vichy and Contrexeville freely, and a moderate mixed diet free from meat and meat extractives and containing a minimum amount of protein. Glycerine in doses of drs. 1-4 has been recommended.

**Tuberculous Kidney.**—Tuberculosis produces 3 types of renal disease :— (1) Caseation and the formation of cavities. (2) Scattered nodules throughout the kidney. (3) Primary ulceration of the apices of the papillæ.

Hæmaturia may be the first symptom, and marked in early stages without much pain. It is most at night. It may only be discoverable by microscopical examination. In later stages it is often slight or absent. Micturition is frequent, if the bladder is involved, and vesical tenesmus common. Incontinence and dysuria occur in the younger children.

Pus is found early, and the urine is acid if the pus is not excessive. Unless the ureter is occluded the urine contains much albumin, casts, pus, lymphocytes and red cells. Bacilli can be found on examination of the centrifugalised deposit, and may be present in the absence of pyuria. The smegma bacillus must not be mistaken for that of tuberculosis. Inoculation of guinea pigs affords confirmation, but takes 5 weeks.

Pain is dull, aching, continuous, without exacerbations and not increased by exercise, and chiefly felt in the loin or reflected therefrom. At the disease progresses the symptoms are anorexia, furred tongue, sweating, frequent pulse, emaciation and hectic fever. A lump is felt in the loin, and per abdomen if there is much wasting. The disease spreads down the ureter to the bladder, modifying the symptoms. Cystoscopic examination may show changes at the ureteric orifices. The second kidney is affected late or not at all. The urine must be segregated, to ascertain if one kidney only is diseased.



It must be diagnosed from calculus, pyelonephritis, nephritis and tumours. "Pain in excess of pus indicates calculus, pus in excess of pain tuberculous pyelitis" (Clement Lucas). The treatment is that of tuberculosis and removal of the kidney, if the disease is unilateral.

**Renal Tumours.**—Most of these are congenital in origin. Birch-Hirschfeld (1898) called them "embryonal adeno-sarcomata." They contain a mixture of epithelial, adenomatous and connective tissue elements; frequently striped and unstriped muscle fibres, occasionally cartilage and bone; and grow in a markedly embryonal way. Various structures may preponderate in different parts of the same tumour. They originate in the remains of the Wolffian body or from an inclusion of neighbouring structures in the capsule of the embryonic kidney. Frequently they are described as sarcoma, adeno-, lympho-, myxo-, fibro-myxo-, round or spindle celled sarcoma, and some are hypernephromata. A few are secondary growths. Papilloma of the kidney has been recorded.

They are more common on the left side. Usually they are extra-renal and intra-capsular. They surround or displace rather than invade the kidney, which becomes flattened. Some of them infiltrate the kidney. A few may arise from the cortex and even from the pelvis; a few are retro-peritoneal or commence in the adrenals.

Both sexes are about equally liable, the males a little in excess. It is most frequent in the second year of life and most of the patients are under 5 years. Out of Steffen's 219 collected cases 168 were under 5 years, and of these 34 were under 1 and 55 in the second year. Of Senator's 96 cases under 10 years 55 per cent. were under 2 and 85 per cent. under 5 years. Of Starr's 54 cases 44 were under 5 years. Other collections show a similar preponderance in the early years of life.

The tumour grows rapidly but produces no constitutional symptoms during the first half of its course. Later, great and rapid constitutional disturbance sets in. The growth may become so enormous that one might speak of the case as a tumour with a baby attached. Abbé removed a 7-lb. tumour from a 13-month old child who only weighed 15 lbs. after the operation. The child was in perfect health 3 years later. In the Middlesex Hospital Museum is one weighing 31 lbs., removed from an 8-year old boy.

*Symptoms.*—There is rarely much pain or marked urinary symptoms. Hæmaturia is the first sign in about 10 per cent., usually microscopical in amount. It may occur early or late, once or frequently, be scanty or abundant, and intermittent. Occasionally it is profuse, forms clots in the ureter or bladder, and causes pain. Wentworth states that it was present in 85 out of 232 cases (36 per cent.). A tumour was noted in over half his cases. Pseudo-fluctuation is often present, sometimes pulsation and a souffle. The presence of resonance, due to the colon, between the upper part of the tumour and the liver or spleen is of great diagnostic value. The outline is smooth and rounded; and the tumour is movable, grows forward



and does not bulge in the lumbar region. Either hæmaturia or a tumour may occur alone or conjointly; or both may be absent, in which case the disease runs a latent course and cannot be diagnosed. Pyelitis and pus cells, hyaline and granular casts have occasionally been observed. Pain is inconstant. In the early stages the child looks and feels quite healthy. The tumour is found accidentally or attention may be attracted by hæmaturia. Abdominal enlargement is often the first sign. Subsequently anorexia, digestive disturbances, dyspnœa from pressure, ascites, and fever up to 104° F. develop; sometimes diarrhœa, retention or frequent micturition. Cachexia and emaciation are late symptoms.

The mean duration of unoperated cases is 8 months, of those operated on it is 16-17 months (G. Walker, 1897). It varies between 10 weeks and 1 year from the date of diagnosis. The older the child, the slower is the growth. Death results from exhaustion or pressure effects. The ureters are rarely obstructed. Calculi are not infrequently found in the renal pelvis. The opposite kidney undergoes hypertrophy and is often affected with chronic interstitial nephritis. Metastatic growths are a rare and late development, and occur in the lungs, renal veins, vena cava inferior, lymphatic glands and liver in order of frequency.

The tumour must be diagnosed from tuberculous peritonitis in which the matting together of the intestines may simulate a new growth. I have known such a case explored. It may be mistaken for tuberculous glands, enlarged spleen, tumour of some other organ, hydronephrosis or a cyst. The semi-fluctuation over a solid tumour is a source of error.

*Treatment.*—The only hope of cure is early removal. The prognosis then depends on the nature of the growth and its complete removal. Of Walker's 148 collected cases 74 were operated on; 27 died from the operation, 28 from early recurrence, 15 recovered but the subsequent history was incomplete, and 4 were well when seen 3 years later. Steffen states that 18 out of 88 cases were permanently cured. Others regard the outlook as still more hopeless. Not one of 21 cases was cured (Bland Sutton).



## CHAPTER XLVII.

### THE GENITAL SYSTEM.

*Affections of the Male Genitals—Malformations—The Penis—Circumcision—The Urethra—Testes—The Mammæ—The Female Genitals—Vulvo-vaginitis—The Vagina, Uterus and Ovaries—Masturbation—Menstruation—Puberty.*

**Malformations.**—Various deformities and their mode of development have been described in the section on malformations of the rectum (p. 332). The penis is developed from the genital eminence which appears in front of and within the orifice of the cloaca in the fifth or sixth week of foetal life. A solid cord of epithelium is formed and is hollowed out into a groove, the lips of which unite in the eleventh or twelfth week and convert it into the penile or spongy urethra. It is complete about the fifteenth week and the raphe indicates the line of union. In rare instances the urethra, or both the penis and urethra are absent. Volpa (1903) reported a case of double penis, associated with double scrotum, a double bladder with two large intestines opening into it, imperforate anus, one kidney, one ureter and one testis. Malformation of the raphe may occur alone or in conjunction with torsion of the penis, the frænum being on one side of the median line and the meatus placed obliquely.

In *epispadias* the urethra opens on the dorsal surface of the glans or penile portion. Occasionally there is no symphysis and it is associated with incomplete development or exstrophy of the bladder. Incontinence is often present. Do not operate before the seventh year.

In *hypospadias* the urethra opens on the under surface; most frequently at the junction of the glans and frænum. The orifice is gutter-like and may present a meatal dimple; or the glans grooved and the orifice at the corona. In other cases the opening is penile or peni-scrotal. In a third variety it is perineal, and the aspect of the parts simulates the female genitals if the testes are undescended (hermaphroditism). There is no true hermaphrodite, if the testes and ovaries are alone regarded as the sign of sex. A male may have Fallopian tubes and a well-formed uterus, with a narrow vagina opening at the sinus pocularis. Such a uterus is occasionally found in the inguinal canal or scrotum (Bland Sutton). Occasionally the penile urethra is short, though the groove has united, and the penis curves downward.

The *prepuce* is frequently narrow and adherent; rarely imperforate,



hooded, and bifid, or practically absent. It may be so short as to simulate circumcision; or absent on the under surface and without a frænum.

The *urethra* may be stenosed or show complete atresia. The meatus may be absent for the first  $\frac{1}{4}$  in. The urethra may be absent entirely or partially, notably in the glans, or closed by adhesions. The defect produces patent urachus and umbilical fistula, or ectopia vesicæ. The meatus may be congenitally narrow. Cylindrical stricture, membranous duplications of the urethra, and congenital or acquired diverticula are very rare.

*Priapism* is due to local irritation by retained smegma, highly acid urine, undue excitability of the lumbar cord, bad habits, and rarely thrombosis or hæmorrhage into the corpora cavernosa.

**Phimosis and Circumcision.**—True congenital phimosis is uncommon. Often the orifice is constricted and rarely it is absent. Phimosis may be secondary and cicatricial. The actual cause is often a constriction of the mucosa and not of the skin. Constriction may lead to ballooning of the foreskin on micturition. A long and narrow foreskin is characteristic of male babies, while the penis is small and undeveloped. It may be too long and too narrow. The glans and prepuce are adherent by reason of the persistence of the epithelial agglutination of the surfaces. Few babies are born with the adhesions fully separated, but separation takes place in the course of some months. The adult condition is reached by the eighth year. Extreme constriction of the orifice may lead to dilatation of the urethra, bladder, ureters and kidneys, hydronephrosis and pyonephrosis, and atrophy of the renal tissues. Such results are extremely rare. If milder in degree, it may cause retention, accumulation and decomposition of smegma, eczema and balanitis, preputial calculi, adherent prepuce, meatal stricture, enuresis, urethritis, cystitis and pyelitis. In prolonged cases of local irritation the foreskin becomes thickened and non-retractile, leading to attacks of balanitis and difficulty in coitus in later life. The local irritation is supposed to induce restlessness, irritability, screaming attacks, frequent micturition, dysuria, colic, masturbation, insomnia and pavor. The straining to micturate is said to develop or maintain hernia, prolapsus recti and hydrocele. On the whole there is remarkably little evidence that these various affections depend on the cause assigned. They occur independently, and may be absent although the supposed cause is present and well marked. Retained smegma and local irritation are the two most frequent sequels.

However advisable for hygienic reasons or with the idea of diminishing the risk of contracting syphilis, circumcision is by no means always required on anatomical grounds. A long foreskin at birth may be insufficient to cover the glans when the penis is fully developed. If it can be retracted with moderate ease, it should be left as a protection for the sensitive glans. The irritation from retained smegma is no argument in favour of operation, if the prepuce can be retracted, but it is a slur on the care and cleanliness of those responsible for the welfare of the child.



In many babies it is quite sufficient to separate the adhesions with a blunt probe, without causing bleeding. Others can be treated by dilatation of the foreskin with dressing or artery forceps, until it can be easily retracted. It is then cleaned, oiled and replaced. The retraction and oiling is done daily by the attendant, if it can be replaced easily. Otherwise there is the prospect of being summoned hastily to cure a paraphimosis. If the surface bleeds on separation, adhesions are almost certain to recur, for the daily retraction cannot be carried out without pain and is neglected. If simple measures fail, circumcise ; incise the mucous membrane only on each side ; or make a longitudinal dorsal incision through the foreskin. Enlarge the meatus, if it is unduly narrow.

Circumcision is the best operation, though neither trivial nor always harmless. Sepsis, sloughing of the skin and extensive scarring, sloughing and gangrene of the penis, fatal hæmorrhage, erysipelas and pyæmia have ensued. Hæmorrhage is usually due to neglect to tie the vessels of the frænum. Tuberculosis and syphilis have been transmitted, when the operation has been done as a religious rite and not by a trained surgeon. Hæmorrhage is rare in this mode of operating, for the Jews remove the skin only, do not cut the mucous membrane, and carefully avoid the frænum. Then the operator takes wine in his mouth and sucks the penis. Neither ligatures nor sutures are used. Apart from the dangers, the operation may be a source of discredit to the operator and of subsequent trouble to the child. It is by no means rare to find an excessive amount of skin removed ; or to see a chronically thickened preputial stump or a mass of redundant skin giving the penis an unkempt ragged appearance, which spoils the surgeon's reputation for years and is a constant source of gossip among the female members of the family, although the inartistic appearance is eventually lost or forgotten. Great care should be taken not to remove too much skin and to leave enough to cover the corona. Possibly the friction of the exposed sensitive glans against the clothing may lead to masturbation, a habit quite as common among the circumcised as the uncircumcised.

*Paraphimosis* is the result of forcible retraction of a narrow foreskin and constriction of the penis behind the glans, unless it is replaced. It causes pain, œdema, ulceration and gangrene. Cold alum compresses will lessen the congestion in early cases, if they cannot be reduced in the ordinary manner. In others it will be necessary to incise the preputial ring at the site of compression, and to circumcise later.

*Balanitis* or balano-posthitis is a purulent inflammation of the prepuce. It becomes swollen, red, and shows patchy ulceration. The exudation may be profuse, the swelling great, and gangrene may ensue. It gives rise to dysuria, increasing difficulty in micturition, cystitis, hydronephrosis, etc., and even death from uræmia, when neglected. The treatment is on ordinary surgical principles.



*Urethritis* is rare in infants. It is due to trauma, foreign bodies, balanitis, and gonorrhœa. Apert and Froget (1906) reported gonorrhœa at 10 months, with secondary phlegmonous inflammation of the whole posterior half of the scalp. Urethritis causes dysuria and a discharge of mucus, muco-pus or pus. The symptoms and treatment are the same as in adults.

Rare affections are *tuberculosis* and *carcinoma*. Tuberculosis of the glans may be a sequel of circumcision. Stiles reports a case in a child, aged 6, occurring after an interval of 3 years. It may cause stricture or even necessitate amputation. The careful local application of flowers of sulphur may be curative. Carcinoma of the penis has occurred in a 2-year old boy (Creite, 1906). He had persistent priapism for 8 days. The tumour was as big as a walnut. The corpora cavernosa and posterior half of the corpus spongiosum were infiltrated. Medullary carcinoma of the *prostate* may occur in the first 7 years of life and as early as 6 months of age. The inguinal glands are soon affected and metastasis is general. A few cases of sarcoma and myxosarcoma are on record, one of them (Edington, 1909) in the second year of life.

**Testes.**—At birth the testes vary in size from an orange pip to a small nut, and they grow little until puberty. The left one is usually larger than the right. Tri-orchism is rare; the third testis small and atrophied. The vas can be felt as a fine cord and the epididymis is loosely attached. Displacements are due to maldevelopment.

*Undescended Testis.*—In *cryptorchism* the testis on one or both sides remains within the abdomen either just below the kidney, in the foetal position, or in the iliac fossa. *Ectopia testis* is the descent to some abnormal position such as the perineal, scroto-femoral, femoral or penile regions. Partial descent, or *retentio inguinalis*, is the most common type, the organ remaining in the inguinal canal, about the groin, or in the upper part of the scrotum. In these situations it is small, soft, movable, and liable to be mistaken for inguinal hernia. Complete descent may be established in later life, up to 21 years of age.

Non-descent causes failure or arrest of development. Fibrotic changes, due to chronic inflammation from trauma or strangulation, render the organ useless. Semen is absent in bilateral cases, and is absent from the vesicula seminalis on the affected side in unilateral ones. Sections show fibrosis and atrophy, and absence of marked development of the tubules at puberty. Imperfect development is likely to interfere with perfect development of masculine characteristics. The skin remains soft and white, stature tall and narrow, muscles poor, and larynx small. Intelligence is defective, temper good, courage small, and the letter R is pronounced badly.

In inguinal and allied types of partial descent a hernia is present in 80 per cent. The gland is liable to injury in all extra-abdominal situations.



If mistaken for hernia the pressure of a truss may set up inflammation, with swelling, tenderness, pain and vomiting; a condition which may be wrongly diagnosed as strangulated hernia. Torsion of the cord may occur. Tumour formation is said to be more frequent in retained testis than the undescended organ, but the evidence in favour of this is slight. Of 78 cases, 4 under 12 years, at the London Hospital in 20 years (Russell Howard, 1907) the testis was undescended in 12, fully descended 48, not noted 18. Atrophy and fibrosis render it less liable to orchitis and tuberculous disease.

*Treatment.*—An attempt must be made to bring down a partially descended testis into the scrotum by manipulation. The mother must be taught to press it down toward the bottom of the scrotum several times a day and hold it in position for a few minutes. Fair success is achieved in infancy. If this fails, operate after the age of 2 and before 5 years of age. Some recommend that operation should be postponed until puberty. This is inadvisable for fibrosis may occur quite early. It is justifiable to wait until the eighth year, if there is no hernia and the organ can be preserved from injury, for the gland may reach the scrotum unaided. Sutures, splints and mechanical appliances are of little value. The organ must not be sacrificed without due consideration, because of the functional effects of its internal secretion. It can be transplanted into the scrotum and inserted between the scrotal fascia and the skin. In 25 out of 29 cases of scrotal placement the ultimate results were unfavourable (L. B. Rawlings, 1907), for the testis became pubo-scrotal, pubic or inguinal, and atrophic. Increase in size may be followed by atrophy. It may succeed under 7 years of age. An inguinal testis is a source of discomfort and very liable to injury and torsion. It is generally useless and should be removed, if unilateral. The remaining organ is sufficient for all purposes. The associated hernia must be dealt with at the same time. It is useless to attempt to save the testis if the epididymis is widely separated from it, if the vessels of the cord have to be severed in transplanting, if the cord is short and the organ fibrotic, and if the boy is over 10 years of age. Reposition in the abdomen is valueless, for the organ does not develop any better in that position. If both organs are in the inguinal canal they are best left alone, unless they become troublesome. Scrotal placement may be tried. Reposition in the abdomen and extra-peritoneal fixation, if done early in life, may possibly enable them to develop at puberty and become functional for a short time, so that the child does not become as a eunuch.

*Torsion of the Spermatic Cord* is rare, about 40 cases on record. It may occur at birth, in the early months of life or later. The testis may be in the abdomen, imperfectly or fully descended, or one which has been replaced in the abdomen. Often no exciting cause can be found. The onset is sudden, sometimes in sleep. It causes painful swelling of the testis, bloody hydrocele, suppuration, gangrene and atrophy. Suppuration is not



common and complete atrophy not invariable. There is considerable vomiting and the symptoms suggest strangulated hernia, but the bowels act with enemata and flatus is passed. Recurrent attacks have been noted.

Try and untwist the cord, if the testis is in the scrotum and the child is seen early. Prescribe rest, elevation of the organ, and morphia for the relief of pain; and tap the hydrocele. Do not remove the organ unless the symptoms are very urgent, or there is hernia with undescended testis. Strangulation may occur from pressure on the cord.

*Acute Orchitis* is due to injury, gonorrhœa, other infections, rarely infarction, and mumps (q.v.). The testis is swollen, very painful, and the overlying skin is inflamed. It must be elevated and supported, an icebag applied, the bowels kept open, and the patient in bed.

*Syphilitic Orchitis* is not infrequent in congenital syphilis, though apparently overlooked until it was described by Després in 1875. Still found it present in 8 per cent. of 64 cases and regards it as pathognomonic. It is found in the first 3 months of life and up to 3 years of age. Generally it is bilateral and associated with hydrocele. Usually the testis is much enlarged, hard and painless. It is not necessarily enlarged and in such cases the diagnosis depends on the feeling of hardness. It never suppurates. The epididymis may be secondarily affected. Microscopically there is a diffuse interstitial cell proliferation; rarely gummatous and causing an irregular outline. Gummata are rare, but may occur in the testis or epididymis. The disease ends in atrophy, not necessarily complete; and may result in infantilism, impotence and sterility. The diagnosis depends on the chronicity, absence of pain, stony hardness, and other evidence of congenital syphilis. Tuberculous testis may occur in this disease. The treatment is that of the cause.

*Tuberculous Testis* is not very rare. It is really an epididymitis. A congenital case has been reported by Dreschfield (1884), and one at a few days of age by Giralaldès. It is most common under 2 years of age, unilateral, and more frequent on the left side. Generally it is secondary. Injury is a predisposing factor. Hydrocele is present in one-third of the cases. It may begin insidiously, or come on acutely and present a slight urethral discharge. It may remain quite localised. The tendency in babies is to early abscess formation but without fistulæ. It is treated on general principles, and by removal if suppuration ensues.

*Tumours* of the testis are clinically malignant. Innocent ones are very rare; so, too, teratomata, either dermoids or sebaceous cysts, in the testis, scrotum or inguinal canal. Ten per cent. of malignant tumours occur under 10 years of age. Not one out of 114 cases (Kobert) was between 10 and 15 years old. Some are congenital. Of 16 cases in 1932 tuberculous children 1 was noted at one and 2 at two months (Demme). Of 16 cases in 5566 children 6 were in the first and 6 in the second year (Jullien). It is a trifle more frequent on the right side and occasionally bilateral; right 47,



left 41, bilateral 5 (Kobert). Injury and undescended testis are the chief predisposing causes. Many are embryomata, derivatives of the three embryonic layers and analogous to certain ovarian tumours. Others are really columnar or spheroidal-celled carcinomata, beginning in the tubules of the testis or vas. Chondrification, mucoid degeneration, hæmorrhage and cyst formation are liable to occur. Fibro-cystic tumours, containing cartilage, are rare in infants. A few are sarcomata. Usually the tumour is soft and fleshy. D'Arcy Power (1907) states that sarcoma occurs in boys of 10 years and over; and that it usually starts in the epididymis.

The swelling is gradual and painless. Testicular sensation varies as the amount of testis left and is lost early. Dull, aching or lancinating pain is caused by rapid growth. Dragging pain in the groins and pain across the loins may be felt. The testis retains its shape, like orchitis, and the growth is smooth until the tunica albuginea is perforated by bosses. It is firm, heavy and hard at first. Later it becomes soft, elastic and fluctuating, like a cyst. It may be masked by hydrocele or hæmatocele. The epididymis is stretched and flattened. The scrotum may become adherent and the growth fungate in late stages. The prognosis is grave and generally fatal. Secondary growths often occur in the lumbar glands and may press on the vena cava inferior and on the portal vein, causing œdema, ascites and dilated superficial abdominal veins. Deposits in the cord are rare. The peritoneum may be diffusely infiltrated. The only hope of cure is early removal of the tumour. The embryomata are not always malignant. One tissue only may become malignant, and simulate sarcoma or carcinoma. Sarcomata are distributed by blood vessels and are more malignant than the carcinomata which are disseminated by lymphatics.

*Hydrocele* is congenital or acquired. The former type has been described (p. 328). Translucency is of no value in diagnosing it from a congenital hernia. The acquired type is rare and like that of adults, but does not attain a great size. It is due to injury, or secondary to disease of the testis or epididymis. It may simulate hernia or an enlarged testis. Simple puncture with sterile needles in several places may cure it. Operation and excision of the sac is rarely needed. A *hæmatocele* is still more rare, and due to trauma or torsion of the cord.

**The Mammæ.**—*Amastia*, or absence of the glands, is very rare. *Polymastia* is not very uncommon, especially among the Japanese, being present in women 5.19 and in men 1.6 per cent. (Teizo Iwai, 1907). The statue of Diana of the Ephesians in the National Museum at Naples has multiple mammæ. Polymastia may be atavistic (p. 17). Normally many glands are developed bilaterally along the abdomen, and in the human female all but two atrophy. Occasionally it is a spontaneous variation, or the result of dichotomy of a single gland. The peculiarity runs in families and is transmitted by females. The accessory glands may be on any part of the trunk, commonly in the pectoral region, and have been seen on the



arms, thighs, cheeks, and even the vulva. They are rather more frequent on the left side. Rarely more than 3, but as many as 8 have been noted. In structure they vary from a normal gland with a nipple to a mass of connective tissue containing a few acini. The mammary gland is a modified sebaceous gland. Accessory ones situated out of the mid-line of the body are probably not mammary glands but really giant sebaceous ones. Supernumerary glands with nipples may secrete milk. According to Iwai, polymastia is more common among the tuberculous and those with polymastia are more liable to this disease. Supernumerary nipples are not infrequent (polythelia).

*Gynæcomastia*, hypertrophied breasts in the male, represents an atavistic tendency, a reversion to a time when the males aided the females in suckling the young. These glands contain a large amount of fibrous tissue and perhaps as many acini as in the female breast. They may develop chronic mastitis. Humboldt investigated and recorded the case of Francisco Lozano who nourished his infant son at the breast for several months. Similar instances on record are not so well authenticated. Precocious lactation and mammary enlargement have been reported in a male child, 3 months old, with congenital morbus cordis. The breasts were about half the size of a billiard ball, areolæ prominent and well marked, nipples large. The increase in size began at 5-6 weeks of age. The secretion resembled milk rather deficient in fat. There was no evidence of mastitis or history of manipulation of the breasts. The child was large, the penis big, and the anterior fontanelle closed. On section after death the glands were normal secreting organs.

*Mammæ in the Newborn*.—The breasts are almost invariably enlarged. The increase in size begins on the second or third day, attains its maximum in the second week, and disappears in another week or two. A milky secretion, "*witches milk*," can be squeezed out; in composition like colostrum. The secretion may persist for 2-3 months. If the breasts are unduly large, give a purge and pot. iod., and apply belladonna. Such treatment is rarely necessary.

*Mastitis and Mammary Abscess*.—In the first to the third week of life the breast may be found tender, and the skin reddened and œdematous. It is the result, almost invariably, of the pernicious practice known as "breaking the baby's nipple strings." This is based on the superstition that it produces wellformed nipples, whereas it is probably the chief cause of retracted and ill-developed nipples. The nurse manipulates the nipple with the fingers and thumb until a little secretion is squeezed out. This is followed by painful swelling of the breast and abscess formation, if infection is conveyed via the nipple. In addition to the local signs the mischief causes fever, anorexia, vomiting, liquid stools and restlessness. The ultimate results may be bad in girls because of partial destruction of the glandular tissue. The condition must not be confused with that of physiological



enlargement and lactation. *Treatment* is preventive ; the local application of lead lotion ; and incision if pus forms. The incision should be made as near the periphery as possible and in a line with the ducts.

*Mammæ at Puberty.*—The physiological enlargement which takes place at 10-15 years of age may cause painful swelling and irritability of the mammæ. It does not lead to ill results. Active treatment is unnecessary. The breasts must be protected from undue pressure. Sometimes the pain is more or less hysterical in its causation. A similar condition of the breasts may occur in boys at puberty. Angioma, adenoma and other tumours, and tuberculosis of the mammæ are rare diseases in early life.

**The Female Genitals.**—Adhesion of the prepuce of the *clitoris* is probably more frequent in girls than boys, and neglected on grounds of modesty. It usually gives rise to no symptoms ; sometimes to nervous irritability, capricious appetite, poor health, enuresis nocturna, onanism, and possibly epileptiform convulsions and reflex paresis of the lower limbs. Other causes must be carefully excluded before blaming an adherent prepuce for any of these symptoms. I have known ulceration at the base of the clitoris a cause of painful micturition. If local irritation in infants under one year is suspected, the parts must be examined. Adhesions can be separated by a probe but they are liable to recurrence. Circumcision is rarely needed. It must be done under a general anæsthetic and the hæmorrhage controlled by a compress.

The *urethra* and clitoris may show a congenital fissure. The urethra may open abnormally into the vagina or rectum, or present other developmental defects as in the male. *Prolapse* is rare. The everted urethra forms a red, oozing, projecting mass which may be mistaken for a carbuncle. It causes genital irritation, straining, and the discharge of bloody mucus. *Urethritis* is part of a vulvo-vaginitis.

*The Vulva and Vagina.*—Occasional malformations are atresia of the anus and vulva, of the anus and vagina, simple epithelial adhesion of the labia, atresia of the vagina, imperforate hymen, and congenital cysts of the hymen. Some of these are described in connection with malformations of the rectum and anus (p. 333). The bladder may open directly into the vagina. As many as 3 septa have been found in the vagina, exclusive of the hymen. One is not uncommon and is situated about an inch within the canal. The vagina may be occluded by a dense hymen or by a tense membrane immediately above a well-formed hymen. The latter structure may be absent. All these septa depend on incomplete absorption of the tubular downgrowth from which the vagina develops.

*Herpes Vulvæ.*—A herpetic eruption may break out on the mucous membrane of the vulva alone, or in conjunction with a like eruption on the cutaneous surface of the vulva, and perhaps on the perineum and about the anus. The rash and symptoms are similar to those of herpes elsewhere. On the mucous membrane the vesicles break down and form small ulcers,



which may heal in 2-3 days or extend and coalesce into large, superficial, painful ulcers, with pain, itching and discharge. Single vesicles may be mistaken for condylomata, but they are much smaller and more transparent. A diagnosis from vulvo-vaginitis is easily made on inspection of the parts. It is not uncommon for herpetic ulcers to develop on the perineum, thighs and buttocks. The affection is readily cured by cleanliness and the free application of a drying powder, composed of calomel, zinc oxide, talc and starch. Mild ointments must be applied, if ulceration has taken place, spread freely on lint and inserted between the labia to prevent friction. It may be necessary to keep the child in bed for a few days. The youngest case under my observation was 11 months old.

*Noma Vulvæ* is a gangrenous process, similar to cancrum oris and sometimes called gangrenous vulvitis. The chief causes are malnutrition, bad hygiene, and measles. It may follow herpes or catarrhal vulvitis. The bacteriology is the same as in cancrum oris (p. 220). Probably a simple infection is followed by a mixed one. Some cases are due to the diphtheria bacillus, and recovery has ensued under antitoxin treatment. It begins as a slight ulceration of the mucosa with a surrounding area of intense hardness. Or tense brawny induration is first noted, and the mucous membrane is shiny and swollen over the most prominent part. The centre of the swelling becomes dark purplish in colour, and finally gangrenous, breaking down and discharging offensive matter. The induration and subsequent gangrene spread and may involve the whole labium, the mons veneris and the perineum. Such an extensive disease is usually fatal. Milder cases recover, with considerable local deformity from the sloughing, destruction, and secondary cicatrization. Stenosis and atresia of the vagina may result. For details of the treatment and a fuller description of the process reference must be made to the mouth affection.

**Vulvo-vaginitis.**—Vulvitis or vulvo-vaginitis is an inflammation of the vulva and adjacent parts. It may affect the urethra and vagina, and extend to the cervix uteri. It is *catarrhal*, the result of dirt (?), chemical irritation, debility, and traumatism, such as direct injury and masturbation; or *parasitic*, due to saphrophytes in delicate children and after infective disease, oxyurides (?), and pathogenic bacteria, notably the gonococcus. The gonococcal form is more frequent than generally suspected, more dangerous than commonly realised, and sometimes appalling in its results. Leucorrhœa does not occur in healthy children, and, therefore, requires treatment. Normally the vagina contains a bacillus producing lactic acid which prevents the development of staphylococci and streptococci, and to a less extent the gonococcus.

*Simple or Catarrhal Vulvo-vaginitis* includes all cases not of gonococcal origin. It is most common after the first dentition, but may occur in infants. Saprophytic cases are liable to spread from child to child, where many are living under the same conditions, being spread by direct inoculation or by



medium of sponges, towels, thermometers, etc. Masturbation is a doubtful cause, though it may produce a mild degree of local redness and irritability, predisposing to secondary infection. Feeble-minded children may masturbate to a sufficient extent to produce excoriation of the vulva, without setting up vulvo-vaginitis. The discharge consists of epithelial and pus cells, many varieties of bacteria and cocci, often a few diplococci. Cocci are found in normal vaginal secretion. It is as well to regard all cases as infectious, though about one-third are due to non-infective organisms.

*Gonorrhœal Vulvo-vaginitis.*—The mucous membrane of babies is very susceptible to gonococcal infection. Some cases are due to mixed infections by the gonococcus and other diplococci. The disease is most frequent in the newborn and in the first 5 years of life, and may have been in existence for some time before the child comes under observation. It increases in frequency at puberty. Out of 600 cases admitted into the New York Babies' Hospital during one year 70 were affected and 9 others had gonococcal arthritis (Kimball, 1903). The disease is certainly not as prevalent in London Hospitals.

The infection is conveyed by the mother during parturition, by the fingers or infected garments of attendants, from the clothes or fingers of a bedfellow, attempted rape, public baths, bathing water, chamber utensils, sponges, towels, diapers and rectal thermometers. The affection is by no means always due to attempted rape, even in older girls. Undue importance is given to this cause on account of the prevalence of a popular superstition that sexual congress with a virgin will cure gonorrhœa in the male. Nevertheless infection is usually direct, from the fingers, rather than indirect. In most cases it will be found that some other member of the family is affected, usually the mother, and that the liability to infection has been great, apart from sexual causes. The older the child the greater is the possibility of a sexual origin.

The incubation period is about 3 days. In the early stage the *symptoms* are those of simple catarrh, a subacute inflammation of the genitalia often not extending beyond the hymen; with thin yellowish-white discharge, a sensation of burning, pain on micturition, genital itching and eczema, and pain on walking in the more severe attacks. The simple catarrhal variety is usually purulent, seldom very acute, and rarely extends to the vagina, the hymen being a sufficient barrier. In more severe forms the discharge is thicker and dries, forming crusts and causing adhesion of the labia, and is offensive.

In the gonococcal variety there is a thick yellowish or whitish discharge, which on pressure seems to exude from the vagina. The vulva, urethra, hymen and vagina become red, swollen, and painful. Erosions may form and the parts bleed readily. The discharge forms thick crusts on the labia. It is much more apt than the simple type to spread up the vagina and involve the cervix uteri, endometrium, Fallopian tubes and



peritoneum. Painful micturition is by no means constant in either variety and may be absent throughout. Often there are no subjective symptoms, yellow spots on the garments being the only point noted by the mother. Usually there is a slight rise of temperature and a little general malaise, a marked contrast to the effects seen in adults. Prolonged cases cause failure of general health.

*Complications* are less frequent than in adults and of the same character. Extension may take place to the urethra and bladder, but cystitis is rare. Inguinal adenitis (bubo) has been noted. Rectal infection may occur and set up proctitis (p. 338). In babies arthritis and pyæmia are not uncommon. Out of 600 babies 10 had arthritis. One of these had vulvo-vaginitis (Kimball), and 8 developed pyæmia under 3 months of age; and 6 died. He ascribed the infection to gonorrhœal stomatitis. Purulent ophthalmia is the most frequent complication (p. 137). Iritis, apt to be regarded as rheumatic iritis, and endocarditis may occur. Extension of the disease to the uterus and tubes may give rise to salpingitis, pyosalpinx and peritonitis. Many cases of unexplained pelvic disease in virgins might be due to an infantile gonorrhœal salpingitis which has remained quiescent until puberty. Possibly sterility and some instances of deformed or undeveloped uteri, associated with dysmenorrhœa, have a like origin.

*Course.*—The simple type lasts for 1-6 weeks; the gonococcal may persist for as many months or for years, with periods of latency. The prognosis is serious as regards duration, and guarded in view of possible complications.

*Diagnosis.*—The differentiation of the gonococcal from the simple variety depends on bacteriological examination. Take the pus, from the urethra if it is affected, and make smear preparations and cultures. The organisms are found in the polymorphs. They are small diplococci, flattened or slightly concave on their opposed sides, like two little kidneys. They are Gram-negative, form acid in glucose and galactose media, and are readily stained by carbol thionin, pyronine methylene green, and alcoholic methyl violet 2 per cent. solution. Schutz recommends the following differential stain; a saturated aqueous solution of methylene blue with carbolic acid 5 per cent. Stain for 5-10 minutes, wash and decolourise with acetic acid, 5 drops in 20 c.c. water, for 3 secs.; wash, dry and mount. The gonococci are stained blue and other organisms are unstained. The normal diplococci of the vagina are not decolourised by Gram (Heiman). Culturally the gonococcus grows on serum, and on a mixture of hydrocele fluid and agar agar, but not on agar agar alone.

*Treatment.*—The patient must be isolated and care taken to prevent indirect spread of infection. Napkins must be sterilised. These precautions should be taken in even simple cases. The slightest suspicion of conjunctivitis must be at once attended to, as in ophthalmia neonatorum. The instillation of protargol 0·5 per cent. solution into the eyes, and the use of a vulvar pad and T-bandage are preventive measures.



Keep the bowels open. Give quinine, syr. ferri iod., or cod-liver oil; and alkalies, if the urine is highly acid and irritating. Sit the child in a bath or basin, containing milk or water with borax or liq. plumbi subacetat., several times daily; or tannic acid, 1 in 1000, once a day. Dry well and apply with a soft brush or wool, calamine dr.  $1\frac{1}{2}$ , almond oil oz. 2, lime water to oz. 8; or wool soaked in boric acid grs. 20, glycerine dr. 2, liq. plumbi oz.  $\frac{1}{2}$ , fresh milk to oz. 8. It is not advisable to wipe away secretion with mops because of the tenderness; nor to keep lint between the labia, for it prevents the free escape of the discharge. Irrigation is of doubtful value. It attracts too much attention to the parts, may convey infection up higher, and may cause severe pain. It is permissible to syringe away superficial discharge with saturated solution of boric acid, separating the labia. If necessary the lower part of the vagina may be irrigated. Irrigation should be preceded by a hip bath and thorough cleansing with soap and water. Use a douche, held 2 feet above the level of the vaginal orifice, and a soft rubber catheter; and have the thighs flexed and fully abducted. Use saturated boric acid solution, sodium carbonate 5 per cent., permanganate of potash 1 in 1000, ordering a 1 per cent. solution and mixing 4 oz. with 2 pints of water, or 1 in 10,000 bichloride of mercury. Douche twice daily, then blow powdered boric acid into the vagina and vulva, and put on a large pad of antiseptic wool and a T-bandage. Irrigation with sodium carbonate may be followed by the injection of protargol 0.5-2.0 per cent., and a tampon of ichthyol 10 per cent. solution. In gonorrhœal cases douche every 4-6 hours. Apply a simple ointment to the surrounding parts. Bougies of iodoform, or alumnol 2 per cent., and the insufflation of iodoform are useful in early stages. In chronic cases lotions of sulphate or sulphocarbonate of zinc and alum, aa dr. 1 to the pint of water, are required. Or it may be necessary to inject silver nitrate solution, dr. 1-4, beginning with 1 in 2000 and reducing the strength 50 per cent. daily down to 1 in 125 on the fifth day. A 2 per cent. solution may be applied direct to the vaginal mucosa through a speculum every 3 days. The cases are tedious and relapses common, unless the treatment is carried out thoroughly. Packing the vagina may be necessary. Vaccine may be used in chronic cases. No patient is cured until the gonococcus cannot be grown on culture media.

**Hydrocolpos** may result, in infants with imperforate hymen, from excessive secretion of a watery or white viscid fluid. The vagina alone may be dilated, but if the secretion is watery, the uterus is usually dilated as well. The condition has been noted on the day after birth, causing a protrusion like a prolapse at the vulva;  $3\frac{1}{2}$  oz. were let out (Commandeur, 1904). *Hæmatocolpos* and *hæmatosalpynx* may occur after the onset of menstruation. *Tumours* of the vagina are almost always sarcomata. They occur in the very young, and are occasionally congenital. Of 24 collected cases the oldest was only 3 years. Few cases are under 6 months, and it is rare after the seventh year. It begins as a small, flat, sessile growth, or a



smooth convex swelling, in the anterior wall ; spreads very rapidly by continuity ; may involve the cervix uteri, urethra and bladder ; and causes hæmorrhage, disturbed micturition, constipation, and abdominal or pelvic pain. Bleeding is the first sign. The vagina becomes filled with soft, friable, sometimes pedunculated, masses. The chief complications are retention, cystitis, peritonitis, pyæmia, pyelitis, pyelo-nephritis, pyometra and sepsis. The average duration is 12 months. It is almost always fatal, for it is detected too late on account of the absence of early symptoms. Israel, Hollander and Schuchardt report cures after operation. Usually operation is followed by recurrence in 6-8 weeks, and the recurrent growth grows and degenerates more rapidly. Metastases are infrequent.

*Rector vaginal fistula* is due to inflammatory mischief and abscess. Sometimes no cause can be found. In a 7-month old girl the stools had been passed per vaginam with much pain for a week. In a girl, 16 years old, the fistula appeared in the third week of enteric fever. Both cases recovered. The prognosis is always good. *Vulvar abscess*, the result of suppuration of Bartholini's glands, is occasionally seen.

*Uterus and Fallopian Tubes*.—The uterus may be absent or rudimentary, so small as to be even overlooked after death and incapable of palpation during life on bimanual examination. It is a rare condition, except in monsters, and usually associated with absence or deformity of the appendages and vagina, of the bladder, kidneys, clitoris and labia. Cases of apparent absence have been recorded in 2 or 3 sisters, and in cousins, examined during life, with no other defect and no heredity of the peculiarity. Sentiment, puberty, sexual desire and development were normal, except for the absence of menses. *Prolapse* of the uterus and vagina has been reported, e.g., in a case of spina bifida (Burger, 1904). It is due to imperfect development of the pelvic floor and may be congenital. *New growths* of the uterus are rare.

The Fallopian tubes may be affected with pyosalpinx or hæmato-salpinx. Tuberculosis is occasionally seen. It is due to secondary infection from the peritoneum, and almost certainly is never the result of an ascending genital infection. From the tubes it may spread to the uterus.

**Ovaries**.—Complete absence is very rare. Occasionally one is wanting. Exceptionally there are three. The glands rest on the pelvic brim at birth and gradually descend to the sides of the pelvis. Abnormally one may be found in the inguinal canal or even in the labium majus (ovarian hernia). Strangulation may occur in the sac of an inguinal hernia, even in the newborn.

Numerous cases of tumours and cysts are on record. Alban Doran reported double ovarian tumour in a 7-month fœtus. Cystic follicles are not very uncommon in the first year of life and must not be confused with cystic adenomata.

The chief varieties are simple cysts, adenomata, dermoid cysts and sarcomata. Papilliferous cysts and parovarian cysts are unknown or



exceptional. Bland Sutton's statistics of 100 collected cases operated on under 15 years of age, and the operative mortality, are :—

- (1) Simple cysts and adenomata 41 ; deaths 3.
- (2) Dermoid cysts 38 ; deaths 5.
- (3) Sarcomata 21 ; deaths 7.

Howard Kelly (1901) recognises two groups :—(1) Cystic ; adenocystomata, unilocular cysts, dermoids and teratomata. (2) Solid tumours ; sarcomata and carcinomata. He gives the mortality under 4 years, independent of the character of the tumour, as 50 per cent. Of 55 cases of uni- and multi-locular cysts 4 died ; 10 out of 47 dermoids ; and 8 out of 24 solid tumours. Of the 55 cystic cases 51 were over 4 years of age and only one of these died. A good many cases have been recorded since. Some show precocious development (p. 200). Under the age of 3 years the tumour is usually a dermoid or a sarcoma ; after that age, an adenocystoma. These tumours have to be diagnosed from tuberculous peritonitis, cystic kidney, sacculated exudations and hydatids.

*Torsion of Ovarian Pedicle.*—In 25 collected cases of ovarian hernia in the inguinal region, usually on the right-side, the pedicle has been twisted. All were infants in the first few months of life ; one over 6 months. Its effects are the production of a tender swelling, irreducible, and without an impulse on cough. The skin may be inflamed and slightly œdematous. The ovary may be gangrenous. There is no intestinal obstruction and no peritonitis. Vomiting is almost always absent. Torsion of the pedicle within the abdomen, generally in connection with a tumour, causes vomiting, abdominal pain, constipation and tympanites. Operative results are excellent.

**Masturbation.**—*Syn. : Onanism, Manustupration, Thigh-friction.*—True masturbation must be regarded as simply a bad habit, the result of education or local irritation leading to manipulation of the penis or vulvar region. In infants, particularly females, it takes the form of thigh-friction. A neurotic heredity is a predisposing cause but is often absent. The exciting factor is almost invariably local irritation by retained and decomposing smegma. This is apt to occur in boys with long narrow foreskins or partially adherent ones, the smegma collecting in pouches. Irritating vaginal secretions or excessive secretion of smegma and adhesions of the preputium clitoridis, and other causes of pruritus vulvæ, are the main factors in girls. Possibly a few cases depend on reflex irritation from the rectum, bladder or kidneys. In some instances the habit is practised by nurses, generally on boys, as a means of soothing the child. The friction of rough diapers, hard sponges or towels may first start the habit ; or even an excess of irritating dusting powder. In older boys it may originate from friction by tight or ill-fitting knickerbockers, riding a wooden horse, or ordinary horse exercise. The friction of the glans penis, exposed by circumcision, is another cause.



Cases have occurred in babies at the breast, as early as the fourth month, but more commonly begin at 6 months to 2 years of age. As a bad habit it is acquired about puberty, or earlier from evil-companions. It is often overlooked in girl babies, for the nature of the movements is not recognised and they are ascribed to other causes. It takes the form of thigh-friction, the thighs being rubbed together and sometimes crossed, or of wriggling movements of the body, or manipulation of the lower abdominal, iliac or pubic regions. Some make to-and-fro, body-rocking movements, while seated on a chair, or rub the vulvar region against the leg of a chair, etc. In a 4-month old baby the vulva was rubbed against the nurse's breast (Strasser). Rarely, the orgasm is induced by mental suggestion. Sometimes, if the child is prevented from carrying out one kind of movement, she develops another. During each act of indulgence the attention is preoccupied, abstracted from surrounding objects; the face becomes red and injected, and covered with sweat; the eyes glitter, the expression is strained, and at times a grin overspreads the features; and sometimes the child grunts or pants. Visible annoyance is expressed if she is stopped. After a variable period the child sighs and sinks back pale and exhausted, and may fall into a sound sleep. The usual position taken up is on the back or half reclining, sometimes a sitting posture on the floor. In male infants the signs of orgasm are rare. Erection of the penis and exudation of a glairy fluid may occur. At first it only occurs occasionally; then more and more frequently, even 50 times a day, until it is impossible to leave the child unoccupied lest it indulges. It may take place during sleep.

Examination reveals nothing positively indicative of the habit. Erection on slight manipulation is suggestive; so, too, redness and irritation of the genitalia, and enlargement of the clitoris or penis, conditions which are a result or a cause. These children may be apparently healthy. More often they are excitable, neurotic, or show signs of mental backwardness. Many are pale and debilitated. On the whole the habit must be regarded as not conducive to mental debility, and that psychical defect is a cause not a sequel. I have never seen any definite evil results, even in cases which have continued for some years.

At a later period the habit is most common in boys and those mentally defective or highly neurotic, especially about puberty. In them the chief causes are local irritation, constipation, overfeeding, late suppers, alcohol, highly stimulating food, evil education and pernicious literature. There are no definite local signs. These boys may show anæmia, general exhaustion, absent-mindedness and lack of memory. Pallor, dilated pupils, languor, debility, vague pains, capricious appetite, disinclination for work or play, headaches, depression, irritability, shyness, loneliness, morbidness and apathy have also been ascribed to excessive indulgence in the habit. These are symptoms of neurasthenia and may occur quite independently. Sexual neurasthenia often begins at puberty.



*Diagnosis.*—The habit is easily recognised by the description, and not infrequently will be practised by a female infant while under observation. Many cases are wrongly diagnosed as *petit mal* on account of the sequence of spasmodic movements for a few moments and sound sleep.

*Prognosis* is excellent in babies. The cure depends on the surroundings of the child and the amount of care and attention which it receives. The attention must be constant and never waver by day or night. The child must never be left alone, unless asleep. If there is mental impairment, so often present in cases seen in young children beyond the age of infancy and occasionally in the infantile cases, the outlook is bad; but the prognosis is that of the mental condition, not that of the habit which may persist and do little harm, unless extreme. Some of these patients practise the habit in public. A boy, 6 years old, with a family history of insanity, had masturbated for 4 years, by manustupration. He was shy, emotional, untruthful, dyspeptic and thin. Although he had a sense of shame he would masturbate in the presence of other children and adults. The penis was large, the prepuce contracted and partially adherent, and there was much smegma preputii. It is criminal to alarm parents about the supposed evil effects of this habit, and they are very grossly exaggerated by the quacks who prey upon the public.

*Treatment.*—Every possible source of local irritation must be attended to. Circumcision is advisable if the prepuce is narrow and adherent, but care must be taken to leave enough skin to cover the corona. The operation, as sometimes done, is in my opinion a not uncommon cause. The few days rest, and the impossibility of practising the habit until the wound is healed, may permanently break the habit. Clitoridectomy is not permissible. It has been followed by cure but there is no evidence that the result was due to the operation. The source of irritability and gratification is merely transferred to some other part of the genitalia. The most important part of the treatment, after attending to local irritation, is to keep the baby under constant observation and to stop her as soon as she begins the peculiar movements. The thighs can be kept separated by very large napkins, made of non-irritating material, or, if napkins are the supposed source of irritation, by a mechanical appliance, such as a couple of padded leather cup-shaped discs applied to the inner surface of the knees and connected by an iron rod working in ball-and-socket joints. In manustupration the hands must be fixed so that they cannot be used.

Locally, apply to the vulvar region carbolic lotion 1 per cent., lead or lead and opium lotion, or salicylated talc powder. Keep the bowels open, attend to the general health and give tonics, bromides, belladonna, and alkalies. Still recommends the liquid extract of *salix nigra*. Camphor, chloral and *cannabis indica* may be useful.

Similar treatment is necessary for older children. Boys must not be allowed to sit on feather cushions or soft chairs, or sleep on feather beds,



nor have their knickerbockers too warm or too tightly braced. Combination garments are often unsuitable, the penis being subjected to friction through slipping in and out of the opening. Cold baths are valuable for children over 4 years, and under this age the judicious use of cold douching in the lumbar region is beneficial. Let the child have plenty of exercise, except riding and swarming up ropes or poles. Allow little animal food, no alcohol and no late meals. Put to bed only when sleepy and watch the child until asleep. See that he gets up immediately on waking in the morning. Allow no heavy bedclothes.

The habit must be regarded and treated as a disease in infants, and as a vice only when it develops in older children free from mental defect. Such cases must be treated by moral influence as a bad habit, a breach of good manners, and a cause of physical unfitness for games. Keep the child's thoughts away from it by providing plenty of amusement, constant occupation, and change of surroundings. Above all do not be alarmist in talking about it to neurotic children. The so-called "straight talks" may do more harm than the habit, especially if a religious element is introduced. Punishment is useful, even in early cases, and must not be neglected, if the child is old enough to understand that the habit must not be indulged in. Blistering the penis with iodine liniment is a punitive rather than a curative treatment, and has little to recommend it. Punitive measures must be used with great judgment and moderation. Some cases are good illustrations of the old maxim that "to spare the rod is to spoil the child." Such measures must only be adopted and carried out under medical advice and supervision. As in cases of enuresis they are apt to be overdone and to be abominably cruel.

**Menstruation.**—Abnormal discharge of blood from the vagina is not always precocious menstruation. It may result from injury, foreign bodies, inflammation, tumours, sepsis or various blood states; and may be simulated by bleeding from a prolapsed urethral mucosa or from other portions of the genitalia.

*Fœtal menstruation* is a red vaginal discharge occasionally noted in newborns, usually on the 5th-7th day, and consisting of blood-stained mucus and clots. It lasts for 1-7 days and does not recur. It must not be confused with hæmorrhage from the vagina in sepsis neonatorum. Halban (1904) believes that it is analogous to true menstruation. All the stages from premenstrual hyperæmia up to subepithelial hæmorrhages and the escape of blood into the uterine cavity can be recognised, just as in menstruation of ovarian origin. Changes also occur after the eighth month in the mammary glands, similar to those in the mother's breasts, and the uterus enlarges. The involution of the breast and uterus after birth takes about 3 weeks. Halban states that comparative changes occur in the mammæ and prostate of male infants. He ascribes it to some placental secretion. More probably it is due to some substance transferred



from the mother. Œdema of the vulva may occur from the same cause and be analogous to œdema of the penis and scrotum sometimes seen in boys, even when delivered by Cæsarian section.

*Precocious menstruation* (*menstruatio praecox*) is rarely seen before 18 months of age. It is commonly associated with precocious puberty, viz., hair on the pubes and in axillæ, erectile nipples, large mammæ, fully developed genitals, large and pigmented labia minora, and generally an ovarian or adrenal tumour (p. 200). Occasionally it takes place in the absence of tumour formation; sometimes in imbeciles; and has been reported in a cretin, aged 9 years, with reversion to a more infantile state on taking thyroid extract (Kemble, 1905). Senile changes commence at a much earlier age than normal. The mothers of some of these children have been remarkably fertile.

*Delayed menstruation*.—Normally the catamenia appear at 10-20 years of age; earlier in the well-nourished and in hot climates than in the underfed and in colder regions. The usual age in this country is 14-17 years. *Amenorrhœa* may be due to congenital defects, such as atresia of the vagina, aplasia of the uterus or ovaries; pulmonary stenosis or morbus cordis, leading to delayed development; and functional or constitutional anomalies. A foetal uterus is associated with lack of pubic hair and of mammary enlargement.

**Puberty**.—Sexual maturity depends on race, climate, nutrition, growth and other factors. It occurs earlier in some families than others, earlier in girls than in boys, and is stimulated by alcohol and premature sexual intercourse. It is present before the onset of the catamenia in so far as the possibility of impregnation is existent. Maternity has been reported at 8 years of age. Probably in remote ages females did not menstruate, being impregnated first, and the development of the function is the result of the customs of civilisation postponing impregnation to a more mature age.

Puberty is characterised by enlargement of the thyroid gland, growth of hair on the pubes and axillæ, development of the sexual organs and functions, and mental and psychical changes. In girls the mammæ develop, pelvis enlarges, buttocks and thighs become fatter, respiration more costal, and the larynx enlarges in a vertical direction, the voice occasionally cracking. In boys hair begins to appear on the face, back and chest; the larynx grows chiefly in its transverse diameter and the voice cracks, the thorax increases in breadth and circumference, breathing is more abdominal; and semen appears in the fourteenth year. Growth of bones and muscles is very rapid at this period. It is most marked in girls at 12-15, and in boys at 14-17 years of age, beginning rather earlier in large than in small children. Both physical and psychical development take place by leaps and bounds, rather than by steady progression, and vary much in different children. Overgrowth leads to undue strain on the heart, and perhaps



relatively unequal development of the body, heart and blood vessels. Yet the heart nearly doubles itself in size and the left ventricle may be hypertrophied. Hence arise feeble pulse, palpitations, dilatation and irregularity, shortness of breath, syncopal attacks and vertigo. Nervous palpitations, anorexia, dyspepsia, constipation, inertia, irritability and insomnia are not infrequent for some weeks or months before the first menstruation. Stooping, lateral curvature and genu valgum depend on muscular debility and unnatural attitudes. Vasomotor instability shows itself in blushing, functional albuminuria, angioneuroses, syncope, etc. Anæmia, headache and epistaxis are common. Chlorosis in girls is possibly due to the withdrawal of iron from the blood and its storage in the liver in preparation for maternity. Vague pains and sensations are felt in the breasts and lower abdomen. Acne, styes, frequent urination, and the effects of constipation, the result of shyness and false modesty, are quite common. Boys develop a sense of superiority and girls one of protective duties. Malnutrition and exaltation of the cerebral processes lead to neurasthenia, hysteria and even epilepsy.

The age of puberty or adolescence lasts from 12-18 years, and the vital processes are exalted throughout but chiefly at the periods of most active growth. Important functions arise and develop. The mortality is low but the tendency to disease is high. The development of normal periodicity in girls is of the utmost importance and should take precedence of that of mind and muscle. There is a craving for knowledge of mind and body, and a delight in rhythm, music and singing. Sentiment, self-consciousness, love, religious feeling and ambition arise. It is the age of hopes, ideals and tender sentiments. Yet the ideas are inchoate, the mind lacking in precision and conscious power, there is a liability to exaggeration and excess, and the feelings fluctuate rapidly in character. The birth of imagination leads to reverie, inner absorption, musing, brooding and even illusions. Vivid ideas seem realities to some imaginative children. The critical faculties are defective, yet there is a consciousness of self, over-sensitiveness and self-criticism. This leads to over-assertion and the bumptiousness of youth. The vocabulary is enlarged and words have a more definite meaning. It is the age of folly, of imitation, of dramatic rôles and poses. Even now the general tendency is objective rather than subjective, and training should be manual, gymnastic, in sports and games, and industrial as well as mental and moral. The disorders of this period of life are commonly those of arrest, defect or excessive development of some organ or function. Hence arise hooliganism, perversions, juvenile crime and secret vice. Many of the ailments at this period depend on the mode of life, notably school life, rather than the age itself.

Hysterical symptoms are very common, and include abnormal appetite, pica, fasting, barking cough, stuttering and convulsive disorders. Hysteria is often due to a sexual shock which is brooded over in secret and can be



cured by talking about it. In the mentally defective, a state sometimes first noted at puberty, there may arise all kinds of mental and psychical disorders. Vicious habits may be unduly developed by imitation, bad companionship, and insufficient supervision in the organisation of school life. Excess depends mainly on inherited nervous instability. Excessive dreaming, somnambulism and migraine may occur. Troublesome disorders of menstruation begin. The first period, more often a subsequent one, may be profuse and even fatal. I have known it cause profound anæmia. It should be treated by calcium salts, ergot and bromides. Possibly horse serum may do good. Irregularity at the onset and for some months is frequent and quite unimportant. Signs of imperforate hymen will appear at this age.

*Treatment.*—The diet must be liberal and may err on the side of excess as long as it is limited to simple foods. It should contain an ample supply of calcium for the growing bones, phosphorus for the brain, protein for muscles, carbohydrates for exercise, and fat for heat. A free supply of oxygen is needed for metabolism. Milk, eggs, cheese, fruit and vegetables should be allowed freely. The dress must be arranged so that there is no compression of the neck, chest or abdomen; and the weight be chiefly supported by the hips and shoulders. From 9-10 hours sleep are needed. The work, posture at work, games and exercise should be carefully regulated; guarding against mental and physical overstrain, as in cycling and long distance running. Judicious guidance is of the utmost value in directing the emotional, psychical and mental development. Fortunate is the child, who is under a suitable and sympathetic teacher, for the future may be made and still more easily marred.



## CHAPTER XLVIII.

### THE NERVOUS SYSTEM.

*Peculiarities—Examination—Reflexes and Special Signs of Disease—Cerebrospinal Fluid—Electricity—Classification of Diseases.*

The brain at birth is immature and its convolutions are flattened and badly defined. It weighs from 9-12 oz., being roughly about one-twelfth of the body weight but not directly proportionate thereto. It grows and develops rapidly and at the end of the first year weighs about 30 oz. Up to the seventh year it grows less rapidly and then quite slowly. At all ages the male brain is heavier than the female. In the newborn the frontal lobes are inconspicuous and the Island of Reil is not well differentiated. On section it is much more grey because of the deficiency in myelin. The special centres are hardly defined during the first 2 years of life and cerebral lesions are localised with much greater difficulty than in adults.

The dura mater is peculiar in early life in that it is closely adherent to the skull and can with difficulty be separated from it; thus preventing extravasations between the membranes and the bone. The amount of subarachnoid fluid is relatively larger than in later life. There are no Pacchionian bodies.

The spinal cord measures 14 cm. and extends to the lower extremity of the vertebral canal up to the end of the third month of foetal life. There is no cauda equina. At birth the termination is opposite the body of the third lumbar vertebra and it gradually ascends until in adult life it is opposite the body of the first lumbar vertebra. The vertebral canal grows more rapidly than the spinal cord.

In structure the nervous system differs in its immaturity in early life from that of adults. Neither its nerve centres nor its nerve tracts are fully localised or developed. The spinal cord possesses almost its full complement of myelin at birth but some of the axis cylinders have not acquired their medullary sheaths. The brain stem, cerebellum and large portions of the cerebrum are devoid of myelin, and myelinisation is not complete before the ninth month. Seeing that the higher centres are imperfectly developed and the nerve tracts ill-defined, the lower centres are incompletely controlled. Hence arise defective co-ordination, spasmodic muscular contractions and unchecked reflex excitability. Development is very rapid, and there is great liability to temporary or permanent



disturbance of function and to organic disease. The whole nervous system is peculiarly sensitive, very excitable, and responds with immense energy to all forms of stimulation. It lacks stability and its inhibitory powers are not developed. At birth and for many months after the infant responds freely to all kinds of reflex irritation. The response is irregular in its manifestation and the symptoms, arising from causes which in adults would produce no effect, may be serious and even fatal, as is the case in convulsions due to errors of diet. Convulsions, unconsciousness and many functional disorders may be due to various kinds of simple reflex irritation. Twitchings and delirium are common in fever.

Organic lesions in early life produce a profound effect from the immediate destruction of tissue and from interference with the subsequent development of the damaged parts. Thus embolism, thrombosis or hæmorrhage may hinder the development of the brain and cause paralysis and varying degrees of idiocy. On the other hand damage to the brain in infancy may be recovered from more readily than in adults. For instance, aphasia due to destruction of the speech centre on the left side during the first three or four years of life may be repaired by the education of the speech centre on the opposite side. Aphasia in early infancy is rarely permanent. Bearing in mind the immaturity of the brain it is important that it should not be overstrained or overstimulated. Functional strain, especially when combined with malnutrition, is a fruitful source of functional nervous disorders such as headache, pavor nocturnus, somnambulism, neurasthenia, chorea, and possibly epilepsy and insanity. Alcohol, tea, coffee and other stimulants, and highly seasoned foods are to be avoided during early life, as well as all those forms of mental stimulation included under over-education and the amusements of city life. To these causes are due many of the neurotic, excitable children seen in large towns.

**Examination of the Nervous System.**—The younger the child the greater is the dependence of the doctor upon objective symptoms and his own powers of observation. Once the child is able to talk a certain value can be attached to what it complains of, bearing in mind that children have not learned to give accurate expression to their feelings. There is nearly always exaggeration of all subjective symptoms, especially pain. Attention must be paid to the *family history* for some organic diseases and psychical disorders depend upon heredity. Alcoholism, syphilis, tuberculosis, hysteria, epilepsy or insanity in one or other parent may have an important bearing on the diagnosis.

A *past history* of febrile disorders, specific fevers, rheumatism, sore-throat or injury, may indicate the possibility of meningitis, acute poliomyelitis, peripheral neuritis, embolism and such like diseases. Over-excitement or errors in diet may point to the possibility of severe nerve storms being purely functional. On the other hand many serious and permanent affections, e.g. infantile paralysis and muscular dystrophies,



may come on so insidiously as not to attract the attention of either the parents or the nurse, and it is frequently impossible to ascertain the date of their origin. The presence of rickets and the nature of the environment must be noted.

*Superficial examination* may give valuable information. Thus the lateral decubitus and retraction of the head suggest basal meningitis. Constantly putting the hand to the head or ear may indicate headache or otitis media. Involuntary signs of pain when a limb is moved or absolute flaccidity of one or more limbs, while the others are moved freely, are also suggestive. Cranial and facial peculiarities are sometimes present in cerebral disease. Among these may be mentioned macrocephaly, microcephaly, hydrocephaly, asymmetry, brachycephaly and dolichocephaly, the facial aspect of the Mongolian imbecile, and the fatuous, stupid or blankly pleasant expression of some other forms of imbecility. A large tense fontanelle, or a prematurely closed one, and separation of the sutures may be of great significance.

*Sensory disturbances* are difficult to estimate. In the very young many sensory variations cannot even be recognised. Pain is often exaggerated and may be very vaguely localised and described by intelligent children. It almost invariably signifies disease and in the very young should not be ascribed to neuralgia. Headache must never be disregarded for it is often the first and for some time the only symptom of organic brain disease or febrile disorders, notably typhoid fever. After school age headache, neuralgia or migraine may arise from overwork. A facial neuralgia may be due to a bad tooth. In a boy, 8 years old, it was a troublesome symptom and, although a dental surgeon could find nothing the matter with the teeth, a stopping subsequently became loose and revealed underlying caries. This was attended to and the neuralgia ceased.

Visual disturbance may be unrecognised until the end of the first year or later. Photophobia is obvious. Temporary or permanent amaurosis is shown by the fact that the infant pays no attention to a bright light, will not follow a light or other object held in front of the eyes, does not blink under these conditions, and still more so if the pupils do not react to light. Deafness is difficult to determine in infancy. It is suspected when no attention is paid to sudden loud sounds. In testing the hearing care must be taken to exclude vibrations which might attract the attention although the child is deaf. The senses of taste, smell, temperature and muscular sense are so difficult to estimate in infants that it is rare for any information to be gained from a most thorough examination of these functions. The intellectual faculties can only be roughly estimated. An intelligent mother will note whether the infant is more backward than her other children were at the same age, or in comparison with those of her friends. She may note vacancy of expression, clumsiness or inco-ordination of movement, and lack of interest in toys and surroundings. The doctor



may make similar observations but, unless he sees the child on several occasions, it is extremely difficult to ascertain whether the mental condition is imbecile or merely backward.

The *motor functions* are more easily studied. It is easy to recognise partial or complete immobility of a limb. Care must be taken to make sure that the immobility is due to true paralysis and not to local disease, such as epiphysitis, scurvy, etc. It is then necessary to ascertain whether the paralysis is cerebral, spinal or neural in origin, or due to primary muscular dystrophy. The association of paralysis with atrophy or spasticity, the condition of the reflexes, and the electrical reactions are of great value, but electrical reaction is difficult to test in early life. Fibrillary and idiomuscular contraction, tremor, inco-ordination and athetosis must be noted.

**Reflex action** can be tested without difficulty. Certain special signs of nervous irritability must be mentioned.

1. *The Babinski Sign*, sometimes called the plantar or great toe reflex.—Babinski found that in disease of the motor tract dorsal flexion, i.e., extension of the toes especially the great toe, resulted from reflex irritation of the sole of the foot. Normally two reflexes can be obtained on irritating the sole; viz. simple plantar flexion, due to a weak stimulus and ascribed to a cortical reflex; and dorsal flexion of the toes and combined movements of the leg, due to a strong stimulus and ascribed to a spinal reflex. In Babinski's sign dorsal flexion results from a slight stimulus and therefore the spinal reflex is exaggerated. It may be due to increased excitability of the cord by strychnia, or diminished excitability of the cortex as in stupor and bromism; or it may depend upon interruption of the motor tract. The sign does not necessarily indicate disease of the motor tract and it is frequently present if the excitability of the cord is increased. It is constantly present in the newborn for the cord is excitable and the motor tract imperfectly developed. Of babies under 2 years of age the plantar reflex is absent in one-third; in one-fourth there is bilateral dorsal flexion and in one-fourth bilateral plantar flexion. In the remainder the reflex may be absent on one side and cause flexion or extension on the other; or there may be flexion on one side and extension on the other. There is no constant reflex during the first year, and during the second year it is inconstant but approaches the adult type. Dorsal flexion is present in 50 per cent. at the end of the first year. In the third year plantar flexion is found, unless delayed by rickets. The extensor response is common in children during sleep, so the test must be applied while awake. The sign is of no practical value before the third year of life.

2. *Mendel's Reflex* is said to be of more value than Babinski's. Place the foot with its inner surface on a firm basis and percuss the dorsal tendons. The normal response is dorsal flexion of the second to the fifth toes and is



present in functional affections. Plantar flexion occurs in organic disease, except tabes.

3. *Schäfer's Reflex* is an extensor toe response obtained by pressure over the insertion of the gastrocnemius or by pinching the overlying skin. It indicates pyramidal degeneration.

4. *Oppenheimer's Sign*.—Tapping the middle of the posterior surface of the tibia from above downward with a hammer causes plantar flexion of the toes in the healthy and contraction of the extensors of the leg in the spastic.

5. *Brudzinski's Contra-lateral Reflexes* in the lower limbs are of two kinds. On flexing the sound limb forcibly in hemiplegia the paralysed limb executes a movement of extension. This is called the "reciprocal" reflex. In the other type, known as the "identical" reflex, flexion of the sound limb induces flexion instead of extension; and in other cases extension induces extension. The identical reflex is said to be constant in tuberculous and cerebrospinal meningitis, disappearing during recovery. One or other may be found in backward children, Mongols, idiots and Little's disease.

6. *Kernig's Sign* was first described in 1880. With the thigh flexed at a right angle to the trunk the leg cannot be straightened without pain on account of contraction of the flexors. It is ascribed to increased irritability of the lumbar and sacral nerve roots, due to meningeal inflammation. Flexion of the thigh stretches these roots, increases their irritability and causes reflex contraction of the flexors. It is especially apt to occur in meningitis of the cerebrospinal type and has been noted in cerebellar tumour, subdural and intracerebral hæmorrhage, thrombosis of the lateral sinus, and occasionally in other diseases such as enteric fever and uræmia. It is a fairly constant sign in meningitis but its absence does not exclude this diagnosis. It is more often absent in the tuberculous than other varieties of meningitis. It may vary from time to time in the course of the disease, is not associated with increased rigidity elsewhere, and usually persists for some time during convalescence. The sign has been noted in the arms. Possibly it is not a reflex and is due to exaggeration of a normal condition, the effect of hypertonicity of the muscles.

7. *Trousseau's Phenomenon*, or the "median nerve reflex."—Pressure on the internal bicipital groove, or elastic compression of the arm for a few minutes sufficient to cause cyanosis and pain, produces the gynaecological hand in tetany. It is due to pressure on the blood vessels or nerves. In children pronation of the hand and some adduction of the fingers is the usual response. It is a sign of nerve irritability. A contraction of the hand in infants of 6-8 weeks old, induced by pressure on the nerves of the brachial plexus, has been described by Hochsinger as the "*phenomenon of the fist*," or "*myotonia of the newborn*." In a marked case flexor or



extensor spasm is persistent, varying in intensity and lasting for a few days to several weeks. Usually the child "makes a fist." It may simulate the tetanoid position. Apparently it is of the same nature as Trousseau's sign, and occurs in gastro-intestinal auto-intoxication, severe nutritional disturbance and tetany in the first few months of life. But it is not associated with the other signs of nerve irritability. Probably it is a mild type of tetany.

8. *Chvostek's Sign*, or the Facial Phenomenon, is an exaggerated excitability of the facial nerve and muscles. A slight touch, tap or pressure on the seventh nerve in front of the ear, or a tap on the face below the malar bone, causes rapid contraction of the eyelids and mouth and twitching of the side of the face. Other exposed nerves are similarly excitable.

9. *Erb's Sign* is an increased irritability of the nerve, especially the ulnar nerve, and muscles to electrical currents, notably galvanism, Finkelstein asserts that it exists in 30 per cent. of infants fed on cow's milk or whey.

10. *The Phenomenon of the Phrenics* is a name given by Solovieff to rhythmical contractions of the diaphragm, synchronous with the movements of the heart.

**Trophic disturbance** is indicated by wasting of muscles, glossy skin, bed-sores, pigmentation and skin eruptions, disorders of the nails and hair, and ulceration of the cornea. Variations in surface temperature, coldness of paralysed limbs, liability to chilblains, and patchy redness or lividity of the affected limb are partly trophic, partly vasomotor in origin.

**Cerebrospinal Fluid** is a true secretion, probably from the choroid plexus. It passes through the foramen of Majendie into the subarachnoid space. It is obtained from the ventricles after death, and during life by tapping the vertebral canal in the lumbar region. Examination of the fluid is valuable in diagnosis. *Lumbar puncture* was introduced by Quinke in 1891 for the relief of chronic hydrocephalus. It had been utilised by Essex Wynter at the Middlesex Hospital in 1889. It is almost free from risk, if properly carried out. Ossipow (1901) collected 12 fatal cases. Headache, nausea and vomiting, giddiness, palpitations, hæmorrhage into the canal and sudden death have been observed. Although I have used the method in a large number of children I have never seen any bad symptom. The patient should be kept quiet for 24 hours afterwards, so the operation is not available for out-patients.

The strictest antiseptic precautions must be adopted. Place the child on the right side with the knees and thighs well flexed, the head and trunk bent forward and the left shoulder depressed, in order to separate the intervertebral spaces as widely as possible; or in the sitting posture with the head and trunk bent forward. An anæsthetic is rarely needed. If required, give a whiff of chloroform or apply the ethyl chloride spray or



cocaine locally. Use an antitoxin or small exploring needle, about 5 cm. long, attached to a syringe as a handle. Cleanse the site of puncture thoroughly. Insert the needle vertically to the surface, 1 cm. from the middle line in the space between the second and third, or the third and fourth lumbar vertebræ opposite the upper edge of the lower spinous process, and direct it slightly upward and inward. Or the median line may be chosen and the needle inserted between the third and fourth lumbar vertebræ, through the interpeduncular ligament which is soft and yielding in children. In older subjects the ligamenta subflava are thick and tough, like indiarubber, and resistant. The depth of the canal from the surface varies with the age and nutrition of the child. In babies the needle enters the subarachnoid space at a depth of 2-3 cm. If it is pushed in too far, it may wound the plexus of veins on the anterior portion of the dura mater. The fluid flows out drop by drop, in a gentle stream or in spurts, according to the pressure. Suction is unnecessary, and aspiration may cause sudden reduction in pressure and unpleasant symptoms. Keep the child still, for movement may break the needle or cause bleeding. Remove the needle, if the pulse fails or the child turns faint. Collect 10-15 c.c. of fluid in a sterile test tube and plug with sterile wool. From a few drops to 4 oz. may be drawn off. It is rarely advisable to remove more than 2 oz. at a time.

Make cultures on blood serum, serum agar and agar agar. Inoculate guinea pigs as soon as possible, or fibrin formation may interfere. Centrifugalise the remainder, or stand it for several hours, and make films from the deposit. Stain and examine microscopically. Estimate the quantity of albumin.

*Normal Fluid* is clear, colourless, faintly alkaline and sterile. Its specific gravity is 1003-1008. It is free from morphological elements, except a few lymphocytes on centrifugalisation, and contains no ferment or fibrinogen. It contains chlorides, traces of carbonates, phosphates, urea and cholin, and of a glucose-like substance, which reduces Fehling's solution and has been definitely shown by Halliburton to be sugar. Serum globulin or albumin is present in amounts of 0.01-0.1 per cent.

*Abnormal Fluid* may be clear, turbid, opalescent, purulent or bloody. Blood is due to injury of the venous plexus, or to bleeding into the ventricles or the subdural space. It may be deficient in albumin in chronic hydrocephalus or contain an excess in inflammatory disease. As a general principle the presence of a mere trace of albumin negatives acute inflammation and a distinct increase in the amount excludes simple hydrocephalus. Coagulation is a sign of inflammation. In tuberculous meningitis the fluid is clear or cloudy, sometimes opalescent, sugar is absent and a fibrin network slowly forms. The albumin is 0.15-0.2 per cent. (Reiken). Cells are occasionally absent. More commonly there is a lymphocytosis, varying in degree. In a few cases I have found an excess of polymorphs; possibly a



sign of severity or of secondary infection. In cerebrospinal meningitis the fluid may be clear at first in the meningococcal type and turbid, containing pus, in later stages. In other varieties it is generally turbid, opaque, yellow or greenish, perhaps pure pus. Its characters may vary in the course of the disease. Sugar is diminished or absent. Albumin reaches 0.2-0.4 per cent. The percentage of polymorphs reaches 80-90 per cent. In chronic meningococcal cases, at the onset or in the course of the disease, the cells may be chiefly lymphocytes. Sometimes only a few are found. Cells are absent in icterus neonatorum, acute phthisis, typhoid fever and dropsical effusions.

*Bacteriology.*—Negative results are of little value. Tubercle bacilli, streptococcus pyogenes, staphylococci, meningococci, pneumococci, b. coli and b. typhosus have been found. On careful examination the tubercle bacillus will be found in most cases of tuberculous meningitis, by staining the fine clot or deposit.

*Pressure* can be measured by Eve's cerebrospinal manometer, but there are many sources of error and the rate of flow affords quite sufficient information. Sometimes fluid cannot be obtained for the needle becomes blocked. Or it may be found on one occasion and not on another, the foramen of Majendie being obstructed and the fluid confined to the ventricles.

*Therapeutically* lumbar puncture is adopted for the relief of pressure, 1-4 oz. being removed at a time. In chronic hydrocephalus it has proved of temporary benefit but the fluid re-collects. It may lessen headache, coma and the other effects of pressure in meningitis, and possibly be of use by removing microbes and toxins. It induces passive hyperæmia of the cerebrospinal vessels, if much is removed, and such hyperæmia may be beneficial in tetany. Its curative value is doubtful. The injection of fluids and drugs into the vertebral canal in the treatment of disease is merely in the experimental stage.

**Electrical Currents** are used for diagnosis, prognosis and treatment; generally the Galvanic, continuous or constant current, and the Faradic, induced or interrupted current. Static electricity and the sinusoidal current are of little use. Galvanism causes a single contraction when the current is suddenly run into or cut off from the muscle; i.e. suddenly interrupting the current by opening the circuit, or allowing it to flow in by closing the circuit. A continuous current causes no contraction unless the strength is suddenly changed. Faradism causes continuous contraction for it is equivalent to a constant make and break of the current.

The current is obtained from the dry cells of Leclanché or from the main. Its strength is measured in milliampères, the unit in medicine, by a galvanometer and should not be measured in terms of the number of cells. A rheostat is useful to regulate the strength, and an alternator to enable a combined faradic and galvanic battery to be employed. Electrodes



are of various shape and size, more or less flat, and covered with chamois leather. The larger the electrode the smaller is the resistance of the body. This resistance is usually high in children and is chiefly in the epidermis. The conductive power of the skin is increased by moisture, so the electrodes are moistened with salt solution before use. The anode or positive pole is recognised by its action on litmus, turning it blue. If the poles are placed in water more bubbles of hydrogen appear round the kathode; if placed in a solution of starch and pot. iod., a deep blue colour will develop round the anode. The kathode is the more active pole when applied to a nerve. This is represented by the formula  $K C C > A C C$ . A knowledge of motor points is essential and will be found in works on medical electricity.

*Testing.*—Get the child used to the sight and noise of the battery, and the feel of the electrodes, before the current is turned on. Confidence must be established. Use no current stronger than necessary to cause contraction, so begin with the weakest interrupted current. Apply a large flat electrode to the back and the testing electrode to the different motor points. Compare the normal reactions with the abnormal, and remember that excitability varies in different children. Then test with the continuous current, beginning with the kathode.

*Normal Reactions.*—A weak faradic current, applied to the motor point of a peripheral nerve or to a muscle directly, causes equal contraction on both sides. When a weak galvanic current, measured by the galvanometer, is applied to the muscle it may cause one or other of the following reactions. The contraction is short, sharp and quick. The first type is the most common.

1.  $K C C > A C C > A O C > K O C$ .
2.  $K C C > A O C > A C C > K O C$ .
3.  $K C C > A O C = A C C > K O C$ .

The important results are whether the contraction is greater or less with the anode or the kathode on the muscle; that is, under which conditions contraction is obtained most easily.

*The Reaction of Degeneration (R.D.)* is more commonly partial than complete for many muscular fibres may be unaffected. A stronger current is required and weaker contractions are produced than on the sound side. The reactions to galvanism may be the same as in complete R.D. In the complete variety faradic excitability is abolished in both nerve and muscle. The response to galvanism of the motor nerve is abolished. The reaction of the muscle to galvanism is exaggerated, i.e., a contraction is caused by a smaller current. The contraction is slow, sluggish and often delayed; and there is an inversion of the normal formula, thus  $A C C = \text{or} > K C C$ , and later  $K O C = \text{or} > A O C$ . Erb regards a slow and prolonged contraction with a weak current as always present and pathognomonic. The presence of R.D. indicates mischief in the lower motor neuron (cord,



nerve or end plate) but does not enable us to differentiate all diseases of the spino-neural system. It is possible that the prognosis is worse in complete than in partial R.D., but some regard both as of equal importance. Electrical reactions are only of value when considered relatively to other physical signs. R.D. is of relative and not of absolute importance. Muscles may show this reaction without any sign of paralysis, and they may have recovered their functions before there is any improvement in their electrical reactions. The reactions on stimulation of the nerve in partial R.D. are of more value in prognosis than the condition of the muscle.

*Electro-therapeutics.*—Electrical treatment is of decided benefit but its value is greatly over-rated. The good results are partly due to the mental effect, as in various neuroses, and in part to the direct influence on the muscle, retarding the degenerative processes and maintaining nutrition, while the nerve or nerve cells have an opportunity of recovery. It is employed in the form of galvanism or faradism, locally or generally. Both poles are applied over the affected area, or the kathode is applied to this region and a large flat anode to the spine. Begin with a current of 3 ma. for 10-15 minutes daily or on alternate days. An electric bath can be used for a single limb or the whole body. The bath is made of porcelain and 2 large flat copper electrodes are placed in it, or 1 such electrode and a small one which is applied to the affected parts. Use a galvanic or faradic current of a strength of 5-10 ma. for 10-15 minutes in water at 95° F. The hip bath is preferable for paraplegic forms of infantile palsy.

**Classification.**—It is impossible to devise a classification based on locality for nervous diseases are rarely localised. One group may be limited to the *Malformations*. Zappert suggests a division into *Endogenous* and *Exogenous* diseases.

An Endogenous or in-born affection is one which is dependent on an inherited weakness, deficient resisting power or faulty development of the nerve cells or nerve tracts. Gowers speaks of this as *Abiotrophy*. The nervous tissues wear out unduly early. There is a physiological premature senescence, slow and insidious in onset, inevitable and progressive in course, and without retrocession. The child is doomed from the moment of conception. These diseases are independent of external influences and are possibly dependent on consanguinity, alcoholism or various toxæmias in the parents. They are racial and familial, occasionally sporadic. They are not always identical in different families and show family modifications of type. The changes are atrophic or aplasic, without inflammation. Higher cells atrophy and lower tissues hypertrophy. The degeneration is localised in one system. Progressive bulbar palsy, primary cerebrospinal sclerosis, amaurotic family idiocy, Freidreich's ataxia, and muscular dystrophies are the main members of this group.

The Exogenous diseases are those dependent on congenital syphilis and possibly tuberculosis; and on post-natal causes, such as microbial



infections, by the action of toxins or mechanical interference with circulation or nutrition. The organism may be present in the affected part, as in meningitis; present in other parts, as in diphtheria and tetanus; or undiscoverable, as in infantile palsy. In certain conditions no morbid change or organism is found, e.g. tetany, insanity, hysteria. Poisons, such as lead and alcohol, are also causes. It is difficult to bring the effects of traumatism and circulatory affections, the results of embolism, thrombosis or hæmorrhage, into this classification, except in so far as they are exogenous in origin.



## CHAPTER XLIX.

### MALFORMATIONS OF THE NERVOUS SYSTEM.

*Cranial Deformities—Cranial and Cerebrospinal Defects—Spina Bifida—Cerebral Malformations—Macrocephalus—Microcephalus—Hydrocephalus—Porencephalus—Syringomyelia.*

**Oxycephalus.**—*Syn. : Steeple or Tower Skull—Pointed Head—Thürmkopf.*—This is a curious deformity of the skull often associated with other congenital defects and stigmata of degeneration. The skull is dome-shaped or the height of the anterior part is abnormal and the temporal regions bulge. Occasionally it is scaphocephalic. The superior maxilla may be deformed. The cause is obscure. The deformity is present at birth but is rarely extreme. Carpenter has reported cases in 3 children of the same family with other congenital defects. Other children of the same mother were normal. It is generally associated with exophthalmos, proptosis, divergent squint, nystagmus, ocular palsy, and defective vision or blindness in one or both eyes. The eyes may be dislocated forward. The ocular defects are due to the deformity. Optic neuritis or post-neuritic atrophy is usual and begins comparatively early in life. It has been ascribed to stenosis of the optic foramen, but this is usually of normal size. Enslin found the nerve constricted in 3 fatal cases. Other possible causes are pressure on the optic chiasma or optic tracts, altered course and kinking of the nerve, defect in the occipital lobe, premature synostosis and increased intracranial pressure, pachymeningitis, foetal meningitis and osteitis. Some of these children are quite intelligent. Others show mental defect, convulsions in infancy and epilepsy in later life. Death is generally due to intercurrent disease.

A modified form shows defective development of the back of the skull, almost complete absence of the occipital protuberance, dome-shaped forehead, protrusion of the eye-balls, blindness and other defects, though the fundi are normal.

*Hyperostosis Cranii*, or excessive thickness of the skull bones, has been reported by Andrewes (1902) in an imbecile boy, 4 years old. All the membrane bones of the skull, especially the frontal and parietal, and to a less extent the lower jaw, were greatly thickened and consisted of uniform dense cancellous bone. Voelcker has seen a similar case in a rachitic imbecile.



*Abnormal Sutures* due to irregular ossification of the parietal bones are of rare occurrence. In a boy, aged 8 months, the right parietal was divided by an oblique suture into 2 almost equal parts. The left bone consisted of an anterior and posterior half, and the posterior half was subdivided into an upper and lower portion. It had apparently developed from 3 centres of ossification. There are records of 10 similar cases, all unilateral. It gives rise to no symptoms. Both bones may be absent. The chief importance of the presence of irregular sutures is that they may be mistaken for fractures or for linear craniotabes. Cranial depressions have been described in the section on injuries at birth (p. 111).

**Meningocele.**—A cranial meningocele is a protrusion of the membranes of the brain, causing a distensile, translucent, non-pulsating tumour of variable size. On coughing or straining the tumour becomes more tense and bulging, except in those rare cases in which the communication with the cranial cavity is closed. It is due to a failure of development of the posterior part of the skull in consequence of hydrocephalus in foetal life. The orifice is situated in the median line of the occipital bone. If it is very near the foramen magnum it is difficult to diagnose from spina bifida, a cervical meningocele. This is a rare situation for spina bifida. A remarkable case was that of a boy who died at the age of 18 days. Labour was difficult and instrumental. A small meningocele protruded through the frontal bone, rather more to the left than the right of the median line. The protrusion was about the size of half a walnut. It could be reduced, and became tense on crying. There were also present a cephalhæmatoma over the left parietal bone, hæmorrhage between the dura and parietals on each side, craniotabes of the posterior parts of the parietals, a lumbosacral myelomeningocele, double talipes and hydrocephalus, but the central canal of the cord was not dilated. The arches of five vertebræ were deficient.

A meningocele may be treated by the withdrawal of a little fluid, and liberal application of collodion to the sac to induce contraction. Operation is justifiable if the communication is closed.

**Encephalocele** or Meningo-encephalocele is a tumour of variable size, usually in the median line, due to the protrusion of brain and membrane. It pulsates and is not translucent. It is most common in the occipital region, but less common than meningocele. It may be situated anteriorly, especially at the root of the nose, and may involve both orbit and forehead; and is rarely sagittal. The symptoms vary with the size and situation and may be absent. Idiocy, squint, nystagmus, optic atrophy, and other deformities may be present. Like a meningocele it may be so large as to cause the appearance of "double head." It must be diagnosed from simple tumours and dermoid cysts. Small ones can sometimes be replaced. They are compatible with life if small and untreated; but if large, death results from secondary infection. Some cases, chiefly nasal ones, can be



cured by excision. The occipital lobes have been removed without affecting vision or other faculties.

**Spina Bifida** is much more frequent than cerebral protrusion. It is due to, or the cause of, the failure of the medullary groove to unite. The arrest of development affects the mesodermic layer only or involves also the ectoderm and endoderm. Its slightest form is a mere cleft, usually lumbo-sacral, with no protrusion of fluid or tumour. This is known as *Spina Bifida Occulta*. It may be overlaid by a hairy mole with hair several inches long. It is occasionally associated with post-anal dimple, sphincter trouble, perineal anæsthesia and club-foot. A fibro-muscular tumour is present and can sometimes be felt in the spinal canal (Zappert). Incontinence of urine, perforating ulcer, neuralgias and palsies may develop later, as a result of mechanical interference with the growth of the cord through its adhesion to the skin. Such adhesions must be removed. It may not be suspected until late childhood or even adolescence. The other varieties depend upon their constituent structures. Of these the myelomeningocele is the most common and the myelocystocele exceedingly rare.

A *Myelomeningocele*, meningomyelocele or myelocele, is a broad based tumour, usually in the lumbo-sacral region and occasionally cervical or thoracic. It is oval, dark red, vascular, soft, fluctuating and elastic, and does not project much above the surrounding skin. The surface may be ulcerated. Pressure on the tumour causes bulging of the fontanelle. It is due to defective development of the medullary groove with consequent formation of a cleft in the vertebræ, defective closure of the spine and membranes which are wanting on the dorsal aspect, fissure in the cord and sulcus in the skin. The bodies of the vertebræ are intact. The wall consists of skin and dura mater, except for thin membrane at the apex, and may become covered with epidermis by proliferation of the surrounding skin. H. B. Robinson reported a case in which the tumour was adherent to the right side of the spine, just below the transverse mesocolon, and simulated an abdominal cyst.

It is often associated with hydrocephalus, dilated fourth ventricle, hydromyelia and other defects such as umbilical hernia, exomphalos and intestinal fistula, club-foot, absent reflexes, and paralysis of the bladder, rectum, perineal muscles and lower limbs. The paralytic symptoms are generally marked; perhaps limited to the feet and sphincters if the tumour is sacral. The ilio-psoas is unaffected. Other signs are right-angled flexion of the thighs at the hip joint; obliteration of anal folds or funnel-shaped protrusion of the anal region; occasionally prolapse of rectum and uterus; and trophic ulceration of buttocks and genito-anal regions. Ulceration of the wall, rupture, sepsis, purulent meningitis and cystitis are likely to occur. Most cases die within two weeks.

A *Myelocystocele* or syringo-myelocele differs from myelomeningocele in that the cord has been enclosed in the spinal canal. It then protrudes



with the membranes through a fissure in consequence of dilatation of the central canal, and is therefore lined with epithelium like that of the central canal. The cleft is of limited size, frequently a little to the side of the median line and usually sacral. The tumour consists of fluid, epithelium, expanded cord tissue, membranes and skin. At first the skin is normal, but later it becomes excoriated and ulcerated from distension by fluid. The ventral part of the spinal marrow is more or less preserved and the motor tracts are perhaps not interfered with, so there may be no palsy. Hydrocephalus, diastasis of the recti muscles of the abdomen, extrophy of the bladder and club-foot are often present.

*A Spinal Meningocele* is rarely seen as a museum specimen and is consequently said to be very rare; probably it is more frequent, less fatal, and often gets well spontaneously or on treatment.

It is a hernial protrusion, between two arches or through a cleft in the vertebral column, containing fluid and no cord tissue. The dura mater may be defective. It is almost always in the lumbo-sacral region, rarely in the cervical or dorsal. It is often pedunculated, transparent to transmitted light, and covered by normal or altered skin, or partially by a thin membrane which readily ulcerates. It is not reduced by pressure. Occasionally it bulges on crying or pressure on it causes bulging of the fontanelle. It may be as big as the child's head. In the sacral region the nerves of the cauda equina may be present in the wall of the sac. Usually there are no symptoms and no palsy. Other defects, and occasionally defective sensibility and movement, may be present in the lower part of the body. A *myelocystomeningocele* is a combination of meningocele and myelocystocele due to fluid collecting between the pia and arachnoid.

Differential *diagnosis* is often difficult. The cleft in the vertebræ is broad in myelomeningocele. In the other varieties the cleft is very narrow, the skin is usually intact, and functional disturbances are infrequent. The skin over a myelocystocele is thin. A meningocele is almost always sacral and has a small pedicle. A myelocystocele may be present anywhere. If it is low down, it is lumbo-sacral and generally associated with other defects. It is rarely possible to be certain that no part of the cord or nerves is included in the sac. The general diagnosis depends on the presence of a tumour at birth in a certain situation, of a definite shape and consistence, and causing pressure symptoms. *Congenital sacral tumours* may be dermoids, teratoid mixed tumours, or true teratomata arising from a second embryo and small in size up to accessory limbs.

*Treatment.*—Simple tapping is followed by re-accumulation of fluid. It is useful as a palliative, to allow an ulcer to heal before proceeding to excision. Repeated tapping may be followed by leakage, infection and meningitis. Morton's fluid, dr. 1 (iodine 1, pot. iod. 6, glycerine 50 parts) is sometimes injected after allowing about two-thirds of the fluid to escape. It is immediately withdrawn. The puncture is painted over with



collodion and the whole tumour with collodion and iodoform. It may cause fatal collapse at the time and usually produces severe constitutional reaction afterwards. It is probably only curative in cases of meningocele and only advisable in quite inoperable cases. A meningocele may be excised. Nerve bundles in the sac must be returned if possible; failing that, they must be removed. The operation should be deferred, if the child is weakly, until the sixth month of age. Operation is useless in myelomeningocele; justifiable in myelocystocele, for the cord entering the dorsal wall of the tumour is functionless; and may be tried for myelocystomeningocele. Buccheri (1908) reported excellent results from the treatment of 19 out of 21 cases by radical closure of the orifice. They were classified as meningocele 2, myelocystocele 18, myelomeningocele 1; males 9, females 12; age 2 days to 3 years, the majority under 40 days. Mean duration of treatment 10 days; 11 healed by first intention; 3 out of 4 ulcerated ones recovered; 3 died. The patients remained well afterwards. Possibly the varieties were named differently. Operation is contra-indicated by hydrocephalus, a large cleft and insufficient skin to make good flaps, other grave defects, and marked paralysis of the lower limbs and sphincters, especially if associated with talipes. Hydrocephalus may develop after successful operation. The chief dangers are fatal shock and collapse, due to sudden escape of fluid or manipulation of nerves and cord, subsequent leakage and sepsis.

**Sacro-coccygeal Dimples, Sinuses and Cysts.**—In many babies and fewer adults a rounded depression or a longitudinal furrow of variable depth is situated over the coccyx above the anus. A dimple or indentation is the most common form. In about one-tenth of the cases there is an orifice of a fistula or sinus. The skin around and lining the depression is pinkish, more glossy and moister than normal, and adherent to the deeper structures. The moisture may be enough to damp the clothes. The size and depth vary from a faint indentation to a deep pit or sinus several centimetres deep, with a very fine orifice or one so large as to simulate the anus. There may be an epidermoid cyst, entirely cut off or possessing one or more fistulous openings. These cysts are not always in the middle line. They are often composed of several more or less dilated tubules, containing mucoid fluid and lined by a single layer of large columnar cells.

These abnormalities are situated near the tip of the coccyx, in the sacro-coccygeal region, over the lower part of the sacrum, or between the coccyx and sacrum. They are probably vestigial remnants. Epithelium, sebaceous matter and hair are liable to be shut up in the cysts and fistulæ. The dimples are lined with true skin but occasionally the cysts are lined with epithelium, ciliated or like that of the small intestine. No treatment is necessary. If troublesome a cyst or fistula can be dissected out.

**Cerebral Malformations.**—The head is entirely absent in *Acephaly*. In *Anencephaly* there is no calvarium or cerebrum, and the cerebellum is unduly atrophic. It may be present in several children in a family. It



is compatible with survival for some days, and often associated with other defects. In *Cyclops* the calvarium is present and the cerebrum ill-developed, arrested or cystic; and the monster presents a single eye in the median line. The convolutions may be unduly large (*macrogyria*) or small (*microgyria*). Fissures and commissures may be arranged abnormally. The corpus callosum may be absent. *Cerebellar aplasia* is a probable cause of Hereditary Ataxia. *Infantile nuclear atrophy*, unilateral or bilateral, may produce ptosis, ophthalmoplegia, a lack of facial expression, and paralysis and atrophy of the tongue with salivation and absent lachrymal secretion, if the hypoglossal nuclei are the ones affected. *Agenesis corticalis* produces cerebral birth palsy. The agenesis may affect the cells, commissures and nerve tracts.

**Macrocephalus**, or hypertrophy of the brain, is a rare congenital condition. The ventricles are often obliterated, occasionally dilated, and the convolutions are flattened. The skull may be enlarged, and the bones thin and porous. A persistent thymus and aplasia of the adrenals have been found in adults. There may be no symptoms until after the first year of life; or convulsions, deep coma and death. Intelligence is defective.

**Microcephalus**.—A small baby has a small head. In microcephalus the head is unduly small in proportion to the size and age of the child. At the end of the first year of life its circumference is 14-17 ins. instead of the normal 18-19 ins. In shape the head may be normal; or it may be dolichocephalic, the transverse measurement being shortened while the antero-posterior is normal or increased and a prominent ridge is present in the middle line. More often it is brachycephalic, the antero-posterior measurement being shortened and the occipital bone unduly vertical. Or it may taper towards the vertex, and be dome-shaped or roughly triangular in coronal section. The forehead is narrow and presents a median ridge. The fontanelles and sutures are prematurely closed. The brain weighs only 30-35 oz. in the adult. It may be microcephalic, though the skull is of normal size. In one variety cerebral disease and non-development are present; in another no disease is found.

It has been ascribed to premature synostosis of the cranial bones, to lack of intracranial pressure, and to imperfect development of the brain. Possibly more than one factor are concerned in each case. The bulk of the cases are of intra-uterine origin. In some instances there is more or less evidence of a past encephalitis or cerebral sclerosis in infancy which has interfered with the proper growth of the brain. It has remained small and consequently the sutures have closed early. Marasmic states in infancy may lead to early closure of the fontanelles and sutures from delayed cerebral growth. Sometimes the fontanelles and sutures remain unduly open and ossification is delayed. Sclerosis, degenerative processes, hydrocephalus or porencephalus may be found after death.



The *symptoms* vary with the degree of microcephalus, and this depends upon the degree of sclerosis or failure of the development of the brain or of part of the brain. The face is small, eyes prominent and expression lacking. If only the anterior portions of the brain are affected, the mental state is that of microcephalus although the head is little smaller than normal. The child is invariably feeble-minded, dull and apathetic, or cheerful and more or less idiotic, but liable to attacks of temper and other emotional disturbance. Many are subject to fits or to irregular movements of the head, of the type of "head nodding," and exhibit loss of power in the limbs, inco-ordination, frequently rigidity, and sometimes general flaccidity. Typical "mandarin" movements of the head are rarely seen except in microcephalus. The child is long in learning to walk and talk, and may never acquire these functions or only imperfectly. Habits of cleanliness are acquired late, and in rare instances not at all. Nystagmus, squint, and impaired sensibility are sometimes noted. The knee jerks are usually absent. In the worst cases there may be blindness or deafness. Defective or apparently defective sight is due to a mental condition. The fundus is rarely abnormal. Sometimes patches of choroiditis are found, and must be regarded as coincident rather than the cause of the visual defect. The evidence is opposed to the view that the choroiditis is due to congenital syphilis. Microcephalus does not necessarily interfere with bodily development and is compatible with prolonged life. Many children remain small, while others grow well, are healthy and even pretty. A certain amount of improvement takes place in the less serious cases, and with careful training the child may be partially taught.

It is probable that in the following instance the affection was post-natal in origin. The child had digestive disturbance at 3 months, enteric catarrh at 5 months, and septic infection of a vaccine pock at 9 months resulting in a bad arm and illness for 3 weeks. At 11 months she began to have attacks of vacancy with pallor, closed eyes, apnoea and clenched hands, the attacks ending in a crow and resembling laryngospasm. These attacks became very frequent and associated with rapid breathing and rocking movements of the head and trunk. At 18 months they still occurred in conjunction with attacks of screaming, without any apparent cause. The screaming might come on after an attack of vacancy or independently. Nodding or lateral movements of the head and trunk also continued. At 2 years all the above symptoms ceased; and at 3 years the child was definitely imbecile and microcephalic, the circumference of the head being the same as that of a younger sister 18 months old. The child was tall, thin and walked well, but could not talk and the knee jerks were absent. In after years she was transferred to an asylum. This case suggests that it was the result of frontal encephalitis due to sepsis.

Another child, 5 years old, showed nystagmus, head jerking, inability to stand and marked loss of sensation. At 9 months it was noted that he



did not try to use his hands. Nystagmus and head jerking during sleep began at 18 months. At 5 years he was excitable, destructive, and only fairly clean in his habits. He bit his nails and had lost a portion of the terminal phalanges of the left thumb and first finger. The knees were scarred by old burns. Sensation was blunted, for he did not cry when burnt or on biting his fingers sufficiently to make them bleed. The hands were used in a purposive but clumsy fashion. Partial flexion and rigidity of the knees, absent knee jerks, talipes valgus, and inability to stand or walk were also present. He was very nervous, peculiarly frightened by the noise of falling rain, and could talk a little. Probably this was of congenital origin and the deficient sensation may have been due to syringomyelia.

*Treatment.*—Linear craniectomy has been advocated and tried on the assumption that the condition is due to premature synostosis and increased intracranial pressure. There is no proof of any such increase in pressure and the symptoms do not suggest it. A longitudinal opening is made by the removal of bone on one or both sides of the sagittal suture. Under strict antiseptic precautions the danger of operation is slight. Carl Beck of Chicago (1897) collected 70 cases of which 20 were said to have resulted in perfect and lasting improvement. Jacobi collected 33 cases of which 13 died, 14 showed little or no improvement, 2 were more improved, and the results were indefinite in the others. It is unlikely that any benefit can be derived from operation in a genuine case of microcephalus.

Craniectomy has been tried in the treatment of imbecility, aphasia, athetosis, paralysis and sensory defects which have proved incurable by medicinal measures. Decided benefit has ensued in some instances, but it would be absurd to expect improvement in cases of gross defect or atrophy of the brain. Trephining is of value for the reduction of the frequency and severity of epileptic fits, and for optic neuritis, a trephine hole covered by membrane acting as a safety valve for the relief of sudden changes in intracranial pressure.

**Hydrocephalus** or “water on the brain” is congenital or acquired. It is convenient to consider all varieties in the present section, as well as certain of the causes which give rise to them. If the excess of fluid is in the cavities of the brain, the hydrocephalus is called *Internal*; if in the subdural and subarachnoid spaces, it is named *External*. Both kinds may be present simultaneously. The main effects are those of increased intracranial pressure. Normally the cerebrospinal fluid runs from the vertex to the theca spinalis and is apparently absorbed at its lower end. If the fluid is poured out in excess or cannot escape from the ventricles, or is not re-absorbed in sufficient quantity, then the intracranial pressure rises. Hydrocephalus can occur without any sign of increased pressure or enlargement of the head, if we include under the term some of the cases of the congenital variety dependent on cerebral malformation.



*Congenital external hydrocephalus* is commonly due to, and compensatory for, diminution in brain tissue, dependent on gross cerebral defects or atrophy. It is spoken of as *hydrocephalus ex vacuo*, a name applied also to similar cases of the acquired type and sometimes to the enlargement of the ventricles secondary to cerebral atrophy, a true internal hydrocephalus. The importance of this type is small.

**Congenital internal hydrocephalus** is either the result of maldevelopment or secondary to intra-uterine basal meningitis. It may occur in twins or in several children as a family disease. Possibly this depends on a syphilitic taint. Striking cases illustrative of maldevelopment may be quoted. The brain of a boy, 4 months old, was found after death to consist of a medulla oblongata, pons, crura cerebri, optic thalami and cerebellum, all of which were small and ill-developed. The cerebrum was represented by a membranous bag, composed of two dilated ventricles. The aqueduct of Sylvius, where the dilatation ended, was extremely stenosed and at one spot of quite microscopic dimensions. The membrane was of the same thickness as the adult dura mater and did not seem to contain any nerve tissue elements. He came under treatment for vomiting of 9 days duration, drowsiness, occasional screaming and a variable amount of rigidity of the limbs. The head was retracted, the circumference  $16\frac{1}{8}$  ins., and showed extreme craniotabes of all the membrane bones, a large bulging anterior fontanelle, an open posterior one and open sutures. There were rigidity of the neck muscles, general spastic rigidity of the limbs, exaggerated knee jerks, and malnutrition. The pupils were equal and reacted to light. In the course of development the anterior cerebral vesicle is divided into two parts, from the centre of which are developed the cerebral hemispheres with their ventricular hollows, the corpora striata and the olfactory lobes. This portion of the brain is known as the pro-cerebrum or prosencephalon. In the above case there was probably, in early foetal life, excessive formation of fluid in the prosencephalon and interference with its escape. Consequently it became gradually distended into a membranous bag of fluid and the nervous elements did not develop.

In a girl, aged 10 weeks, born at full time and weighing  $3\frac{1}{2}$  lbs., a similar condition was present. The head was large at birth and had increased since, measuring 17 ins. The body-weight had increased to 6 lb. 14 oz. The discs were quite white and merged into the rest of the fundus. All the membrane bones were unossified, except for small isolated patches of bone, chiefly situated in the normal sites of the centres of ossification. A third child, a girl aged 4 months at death, showed a like condition of the membrane bones of the skull. She weighed  $3\frac{1}{2}$  lbs. at birth. At 3 months the head was large and top-heavy, 16 ins. circumference. The child attained a weight of 10 lbs. and a cranial measurement of 18 ins. before death. The discs were indistinguishable from the surrounding fundus and there was extensive old choroiditis. The child cried much, seemed blind and unintelligent, rolled



the eyes during sleep and was unable to support the head, but there was no spasticity. The cerebral condition was as in the first cases, though not quite so extreme. The cortex was almost membranous, except in the occipital region where it was about  $\frac{1}{2}$  in. thick. In a fourth case, a boy aged 6 months at death, there was general rigidity of the back and limbs and retraction of the head, with spasmodic exacerbations. It was said to have come on at the age of 7 weeks. A few pigment spots were present in one fundus. The case appeared one of spastic diplegia. At the autopsy it was found that the frontal and parietal portions of the brain were converted into a bilateral cystic formation, bounded mesially by a wall containing a moderate amount of brain substance. In none of these cases was there any evidence of syphilis as the cause of the error of development in the prosencephalon.

This condition is sometimes called hydromicrocephalus, hydranencephalus and bilateral porencephalus. There may be no enlargement of the head, or both the fluid and head measurements go on increasing if the child lives. The head may remain small from premature ossification and union of the sutures. Congenital hydrocephalus resulting from intra-uterine inflammation is identical with the acquired variety.

*Acquired External Hydrocephalus* is a sequence of meningeal inflammation or new growths. Possibly it is compensatory to the diminution of brain substance in cerebral atrophic states. It has been found in marasmus and pernicious anæmia. The amount of fluid varies from a few ounces to a pint, and may cause enlargement of the head and separation of the sutures, unless they are already firmly united. The brain substance is usually œdematous. Symptoms may be absent or take the form of twitchings, convulsions and collapse. An interesting case was that of a boy, 7 years old, taken ill suddenly with severe headache and screaming. Three days later he was irritable, drowsy and intensely stupid. The head was a little retracted and the knee jerks exaggerated. There was no optic neuritis. Three days later he had severe convulsions, mostly of the right side, with unconsciousness. He was trephined over the right Rolandic area and much cerebrospinal fluid let out, after which the brain began to pulsate. The fits did not recur and, after remaining rather stationary for 2 weeks, he rapidly improved. In another 3 weeks he had a further attack of convulsions and unconsciousness, remaining in this state for 3 or 4 days, when he again recovered. Three months later he was apparently well, but his memory was bad and he always preferred to use the left hand.

**Serous Meningitis or Serous Apoplexy.**—The case just described is probably a true instance of a primary serous meningitis, an inflammation of the pia-arachnoid which may spread to the ventricles. This has been described by Quinke under the title of *meningitis serosa ventriculorum*, and by others as *ventricular serous meningitis* or *acute ependymitis*. In its acute form it may be spoken of as “serous apoplexy”; in its chronic type,



as "acquired internal hydrocephalus." It is doubtful whether it is ever limited to the external membranes or the cavities of the brain. If it is, external and internal hydrocephalus may occur alone or conjointly. The fluid is clear and contains an increased amount of albumin, fibrin and cells, chiefly small lymphocytes.

Serous apoplexy in infancy gives rise to convulsions and high fever; sometimes hyperpyrexia and coma. The pupils are contracted and slight rigidity may be present. It is usually fatal in less than 24 hours. Coma is the chief sign in cases of gradual onset. In older children it produces headache, fever, drowsiness, coma, teeth-grinding, conjugate deviation of the eyes, irregular and rapid pulse, retraction of the abdomen and impaired muscular power. Fever may be absent, slight or inconstant. The vision is markedly affected. Less virulent cases display meningeal symptoms and are sometimes described as *meningismus*. This is a bad name, which should be discarded, for similar symptoms may indicate vascular disturbance or cerebral toxæmia.

The etiology is the same as that of meningitis. Post mortem examination may show nothing except a little increase in fluid, a granular state of the ependyma, an œdema of the pia-arachnoid, or the early acute stage of purulent meningitis. It is diagnosed with difficulty from acute encephalitis and simple basal meningitis, and the diagnosis may remain in doubt even when it has passed into the stage of chronic hydrocephalus. Some cases get well. Others become chronic and present headache, vertigo, optic neuritis, vomiting, ataxia, mental changes, etc.; signs such as are seen in cases of cerebral tumour.

*Acute Ependymitis* may be of the type above described. In a few instances it is due to a pneumococcal or streptococcal infection, and after death pus is found in the ventricles, without meningitis. Turbid fluid is often found in the ventricles, pus rarely. It may result from suppurative inflammation of a spina bifida, rupture of an intracranial abscess, occasionally purulent meningitis, and possibly from primary inflammation of the ependyma. It is rarely uncomplicated and may be secondary. The onset is acute with vomiting, headache, and perhaps convulsions. The fever is persistently high and somewhat hectic in type; the pulse and breathing frequent and irregular. Consciousness is soon lost and there is persistent vomiting, squint, bulging fontanelle, slight head retraction, frequent fits and emaciation. During life it is difficult to distinguish it from meningitis. The chief diagnostic features are the bulging fontanelle, exaggerated knee jerks, absent Kernig's sign, and a high temperature. Death results in 3-6 weeks.

**Acquired Internal Hydrocephalus** is a sequel of ependymitis or serous meningitis, and much more frequently occurs in the course of acute meningitis or as a sequel of basal meningitis of varied causation. Many cases, beginning in the first few months of life and diagnosed as congenital,



are really a sequel of a mild unrecognised attack of basal meningitis. It may be regarded as a symptom of this disease. Occasionally it is due to sinus phlebitis, sclerosis or the pressure of tumours. Its frequent occurrence in tuberculous meningitis has led to this disease being named *acute hydrocephalus*, a title which ought never to be used, for the effusion may be due to many other causes. Used without qualification, hydrocephalus means the effusion of an abnormal amount of fluid into the cavities of the brain, and dilatation of the lateral ventricles, the third and fourth ventricles, the iter a tertio ad quartum ventriculum, and even the central canal of the bulb and upper part of the cord. A few cases have followed successful operation for the cure of spina bifida or encephalocele.

*Morbid Anatomy.*—Congenital cases may exhibit gross cerebral defects, spina bifida and other deformities. Evidence of old basal meningitis is exceptional. It is most common in acquired internal hydrocephalus. A new growth is found in rare cases. The amount of fluid varies from a few ounces to several pints. It is greatest in the congenital cases and those arising in the first few months of life, before the sutures have ossified. In cases secondary to inflammatory disease the amount rarely exceeds half a pint in the first 4 years of life. Its composition is that of normal cerebrospinal fluid. It may be turbid and contain more albumin, if it is of inflammatory origin. In rare instances it is purulent, sometimes as the result of operative interference.

As the fluid accumulates the cerebral tissue becomes compressed and thinned, reduced to a layer only a few millimetres thick, constituting a thin wall of a vast cavity. The convolutions are flattened and eventually all trace of them is lost. The occipital region suffers less than other parts of the cerebrum. The septum lucidum is broken down or reduced to a thin membrane, and all the communications between the ventricles are greatly enlarged. The brain substance left in the cortex, if any, is pale and homogeneous, showing no differentiation into grey and white matter; and microscopical examination shows granular and broken down nerve cells. In the worst cases only the cerebellum, pons and basal ganglia are left, and even the basal ganglia may be almost unrecognisable. In milder degrees the ependyma is normal, or finely granular on the surface and perhaps thickened. The choroid plexuses vary. The external enlargement of the skull is conspicuous for the cranium is proportionate to the effusion. The bones are thin and the sutures widely separated, sometimes even those of the base. The gaps are filled in by Wormian bones in mild cases which recover.

*Pathology.*—There may be a past or comparatively recent history of symptoms indicative of inflammatory mischief at the base of the brain. The actual development of the effusion can be watched in certain cases of basal meningitis. It is due to:—(1) Blockage of the foramen of Munro, aqueduct of Sylvius and the fourth ventricle; (2) Blockage of the foramen



of Majendie and the *aperturæ laterales ventriculi quarti*; (3) Thrombosis of the *venæ Galeni*. If both veins are thrombosed death ensues from cerebral anæmia before hydrocephalus can develop. The foramen of Majendie is often non-existent. If no such explanation is available, the affection may be due to *ependymitis*. This is almost certainly the cause in cases which can be drained by lumbar puncture. Idiopathic effusions are said not to be infrequent in rickets but I have been unable to find any evidence in support of this statement. Some of the supposed acquired cases are really of congenital origin, although there may be no signs of the affection until some months after birth.

The *symptoms* are practically identical in congenital and acquired effusion, except that the former may be conjoined with microcephaly. The condition may be suggested by headache, vomiting, mental impairment and fits, but even these may be absent unless the fluid is actually increasing, and the diagnosis has to be based on the physical signs.

Chief among them is the alteration in the cranium. The earlier the disease begins, the larger the head becomes. Its shape is peculiar, remarkably globular, less often pyramidal. It is increased in height out of proportion to its length, and bulges more in front than behind. The forehead is large, high and prominent, and there is a prominence at the root of the nose. The temporal and parietal bones are pushed outward and diverge as they pass upward. The fontanelles are large, tense, bulging and fluctuating. The sutures are separated, perhaps as much as 2-3 ins. The bones are thinned and crackle on pressure, or the whole cranium may feel like a membrane, with or without isolated patches of bone in it, and may even be translucent. The hair is scanty. The scalp is thin and the superficial veins, dilated in an attempt to increase the collateral circulation, show up as dusky blue cords and feel as if running in definite grooves in the bone. If the child is much wasted, the abnormal size of the head is still more evident. It is often so large and heavy that the child is unable to support it, and it may attain a circumference of 30 ins. The face is triangular and looks abnormally small in contrast to the huge cranium.

The eyes protrude, from flattening of the upper wall of the orbit. The sclerotic above the cornea is often exposed, while the pupils may be partly covered by the lower lids. In the early stages there are no ocular changes. At first the pupils are usually small and equal. Later they may become dilated and immobile. Squint, nystagmus, myopia, hemianopia and amaurosis may develop. The blindness is due to pressure of the dilated third ventricle on the optic tracts and chiasma; and optic atrophy is rarely preceded by papillitis in infancy and early childhood. Hearing remains normal for a long time. Alterations in the sense of smell and taste are difficult to estimate.



Other symptoms depend on the causation, the amount of damage done to the cerebral tissues, the stationary or progressive character of the effusion, and the possibility of skull expansion. In mild secondary cases of small effusion, stationary in character, there may be no signs beyond a slight increase in the size of the head. If the fluid is effused slowly, there are few cerebral symptoms for the brain accommodates itself, especially if the bones yield readily. The mental faculties may be normal, or even above the average, although there is a considerable excess of fluid, provided the skull expands. More commonly the child is placid, slow, lethargic, dull, stupid or even completely imbecile. Somnolence is not infrequent. It is extraordinary how long the mental faculties will remain unimpaired, if the fluid collects slowly and the head can expand, in spite of the cortex becoming thinner and thinner. In some cases development is retarded and the child is unable to support the head until 2-3 years old. Walking and standing are interfered with early. The child often does not learn to walk, and the lower limbs remain small and undeveloped. Paraplegic conditions and rigidity of the legs, imperfect control over the hands and arms, contractures of the hands, and exaggerated reflexes are present in severe cases. The habits of cleanliness are lost or are not acquired. The appetite is good, even voracious, and digestion is good. Pulse, respiration and temperature are normal; occasionally the temperature is persistently subnormal.

*Concealed Hydrocephalus* is an effusion in older children with unyielding sutures; either the persistence of a mild attack in earlier life or secondary and limited in degree. Some of these children have acute symptoms; headache, vomiting and optic neuritis. Intense, paroxysmal headache may cause sudden death. Persistent malnutrition, retarded development, peculiarities of temper, morals or emotions, incontinence, palsies, convulsions and insanity may be noted. In others there are fine tremors of the tongue, lips and hands; slight exophthalmos; weakness of the lower limbs; optic neuritis, and fluctuating facial or ocular palsies. Cases of this type, when a sequel of effusion in earlier life, generally have an enlarged cranium and are often called *Chronic Hydrocephalus*.

Retraction of the head, partly from the weight and partly from basal irritation, inability to support the head, vomiting, irregular fever, twitchings and convulsions may be present, if the fluid is increasing or if there is continued basal irritation. Sudden attacks of headache, vomiting and fever are due to exacerbations of the basal mischief or increased effusion.

*Diagnosis.*—The prominent forehead and square head of rickets are not hydrocephalic although the child is often said to “have a touch of water on the brain.” The chief points in diagnosis are that the head is unduly large for the age and size of the child, that the enlargement is progressive and the sutures are separating, and that intelligence and motor functions are becoming impaired. Rapidity of enlargement is of as much value as



the actual size. An increase in circumference of an inch during a month is practically conclusive. Increased size of the head in infants, and increasing mental disturbance and fits in older children, are signs of increasing effusion. The sutures may separate after apparent closure up to 3, and possibly 4, years of age. Active symptoms are due to progressive basal meningitis or greater effusion. Congenital hydrocephalus is difficult of diagnosis, if the cranium is not enlarged, for the mental incapacity is perhaps unrecognisable, unless profound, under 1 year of age. Optic atrophy and spasticity of the limbs are valuable signs, for they indicate gross cerebral mischief or defect. In doubtful cases the tenseness of the fontanelle, the occurrence of fits, and cyrtometric tracings at intervals of a month are of much assistance.

*Course and Prognosis.*—The effusion may remain stationary in the acquired variety and the signs thereof improve. In a mild case the child may develop physically and mentally, and grow up with merely an unusually large head. Others exhibit motor weakness, blindness and permanent mental deficiency (*paralytic idiocy*). Congenital cases generally die in the first few months from marasmus, convulsions or coma; few live beyond 4-5 years. The duration of life depends on the amount of effusion and the amount of care devoted to the child. Some are stillborn, or it may be necessary to puncture the head to facilitate delivery. The head is rarely large at birth, and the increasing size does not attract attention for 1-3 months. The prognosis is hopeless, if the effusion is progressive or due to a tumour. Spontaneous discharge of the fluid through the nose or into the pharynx has occurred and ended in recovery; or the rapid evacuation may prove fatal. Death results from marasmus, convulsions, intercurrent disease, and exceptionally rupture of the head. It may be ushered in by hebetude, flaccid palsy, absent reflexes, and loss of sphincter control. These children are liable to sudden death, headache and vomiting.

*Treatment.*—It is useless to attempt to prevent effusion by diuretics, purgatives, or moderate pressure with a rubber bandage, applied for a week at a time at intervals of a week. The treatment during this period is that of the meningitis which is the exciting cause. Mercurial inunctions may be beneficial. Operative measures are the only means available for the removal of fluid and even then it rapidly re-accumulates. It is useless and dangerous to tap the ventricle with a trocar and cannula, inserted in the anterior fontanelle  $\frac{1}{2}$ -1 $\frac{1}{2}$  in. from the median line. Fluid has been withdrawn by this means and sterilised air introduced through another cannula at the same time, to avoid the results of sudden reduction of intracranial pressure. The child died. Sourma (1888) recommended exposure of the head to the direct rays of the sun daily for a month, 15-50 minutes at a time. It is unlikely to do harm. Lumbar puncture is the best means of removing fluid, if there is a free communication between the ventricles and the theca spinalis. From 1-3 oz. should be removed once



or twice a week, if there are signs that the fluid is increasing. The sudden removal of large quantities of fluid may cause fits or fatal collapse. In my experience this treatment is only of temporary benefit, merely delaying the progress of the case. It was adopted for effusion in a girl, aged 8 months, with drowsiness, retraction of the head and malnutrition; the sequel of basal meningitis (?) 3 months previously. She was tapped 17 times in 20 weeks, 35 oz. being removed in all. During this period she gained 124 oz. in weight. In spite of the fontanelle being no longer tense, the head continued to increase slowly in size and the child was apparently blind and unintelligent. Other cases have also failed. Yet the treatment might be beneficial and even curative, if adopted in the early stages, for at times the effusion may become stationary and it seems to have a better chance of doing so if the general nutrition of the patient improves. Other surgical measures are trephining through the occipital bone and draining the fourth ventricle, or making a permanent communication between the ventricles and the subarachnoid space. Mercury, as an inunction to the scalp, and iodides are the only drugs likely to be of the least benefit. Convulsions are treated by the usual measures. Up to the present the results of treatment are disappointing. It is important to maintain the general nutrition.

**Porencephalus.**—Under the name of porencephalus may be included all those pathological holes, cavities, hollows or depressions which extend from the cortex towards the centre of the brain and often communicate with the lateral ventricle. The name was given by Heschl (1859) to that deformity of the brain in which there is a communication between the surface and the lateral ventricle. It was restricted by Audry (1888) to cases in which there is actual destruction of brain substance; but the condition may arise from non-development. Cavities filled with pus or blood, tumour or parasitic formations, and anencephalic monsters must be excluded. Ireland (1896) divided it into 2 varieties:—(1) True or Congenital; the deformity dating from the fifth to the seventh month of foetal life; (2) False or Acquired; due to the giving way of the wall of the ventricle after birth, owing to a destructive lesion of the cerebral tissue between the ventricle and the surface of the hemisphere. Such a lesion might occur during foetal life, so it would perhaps be better to divide the cases into those of developmental and those of secondary origin.

**Morbid Anatomy.**—Some cavities are cyst-like, limited by the pia and the ependyma; some are limited by the pia and open into the ventricle; and others open into the ventricle and subarachnoid space. A typical cavity is funnel-shaped, lined by pia mater and contains fluid. The fluid varies in quantity and may be so abundant as to amount to hydrocephalus. It is usually as clear as water, or of a tint from yellow to brown. Occasionally the sides of the cavity are opposed and there is no fluid. The cavity is commonly situated in the portion of the brain supplied by the middle



cerebral artery, viz., the third and ascending frontal, the ascending parietal, the supramarginal and first temporo-sphenoidal convolutions. Less often it is in the part supplied by the anterior or posterior cerebral artery. Audry collected 103 cases and found that in 32 out of 96, in which a good description of the lesion was given, it was in both hemispheres and often symmetrical. In 18 almost the whole of both hemispheres had disappeared. The surrounding convolutions may appear normal, may radiate from the cavity outward, and may exhibit a more or less cicatricial change. The basal ganglia are often smaller on the affected side. Secondary degeneration may follow on the acquired form and failure of development of the pyramidal tracts may result from the congenital variety. A congenital cerebral palsy can occur although the pyramidal tracts are undeveloped at birth. Sometimes there is marked asymmetry or arrested development of the cranial bones.

*Etiology.*—About two-thirds of the cases are of foetal origin. Others develop during the first 2 years of life and more rarely later. It has been ascribed to obliteration of foetal vessels (Klebs), to anæmia of the middle cerebral artery (Kundrat), and to non-development. Kundrat put forward the view that the loss of substance affected definite vascular territories, but the majority of cases anatomically contradict this. The acquired form may be due to embolism, thrombosis, hæmorrhage, encephalitis or external injury. Softening of the cortex and underlying parts can result from embolism or thrombosis of a cerebral artery, and lead to the formation of a communication between the lateral ventricle and the surface of the brain. There is very seldom positive evidence of change in the arteries supplying the affected part of the brain. It is most frequent in the region of the Sylvian fissure. Congenital syphilis, obstetrical causes and marasmus in the newborn are suggested factors with no evidence in their favour.

The *symptoms* vary with the size and situation of the cavity or cavities. When both hemispheres are involved there are usually idiocy, often deaf-mutism, convulsions, paralyses, diplegia, rigidity, contractures and cranial deformity. The palsy is of the cerebral spastic type. A small unilateral cavity may cause no symptoms. More often it is fairly large, affects the motor region, and produces partial or complete hemiplegia of the opposite side. The intelligence in these cases may be unimpaired or merely backward. Convulsions are not infrequent in extensive lesions. Aphasia is rare, except in congenital cases with large cavities. Sensation may be unimpaired, but it cannot be accurately tested when the intellectual faculties are affected.

Thrombosis of the posterior cerebral artery was the assigned cause in an epileptic girl, aged 22, who developed left hemiplegia and convulsions at 3 days of age (Gibson & Turner, 1898). The autopsy showed much atrophy of the right hemisphere, with a large cyst in the posterior portion, and atrophy of the convolutions and the left cerebellar hemisphere. Thrombosis of the middle cerebral artery in the later months of foetal life



was the explanation put forward in the case of a patient, 76 years old, who died from pneumonia during dementia of 16 years duration (D. A. Shirres, 1902). He was born at full time, though the mother nearly aborted after a severe fright in the seventh month of pregnancy. Stiffness of the right arm and fingers were noted at 1 week. He was rather backward intellectually, did not walk until the third year, nor talk until the fourth year, and his right foot was always inclined to drag. He had no fits. He learnt to write with the left hand, married twice, had no children, and his mental faculties failed at 60 years of age. The left hemisphere showed extensive porencephalus, a sausage-shaped area of destruction, the brain substance being replaced by a membranous sac containing clear fluid. The right hemisphere weighed 560 gms., the left 302 gms. There was also atrophy of the optic thalamus, the posterior limb of the internal capsule, the corpora quadrigemina and optic tract, and degenerated pyramidal fibres in the cerebral peduncle, pons and fillet. Possibly the cases described (p. 631) as non-development of the prosencephalon or congenital hydrocephalus might be termed bilateral porencephalus.

*Prognosis.*—The affection is often incompatible with life. Only 3 out of 18 congenital cases lived beyond infancy (Kundrat). Of 95 cases, 16 lived less than a month, 14 died under 2 years, 32 died in the second period of infancy or in adolescence, and 33 lived to adult age (Audry). The outlook depends on the extent of the cavitation. If the cavity is single, small and unilateral, there is no inherent reason why life should be shortened. If the cavities are large and bilateral, the child is practically certain to be a hopeless idiot and to die in infancy. The degree of diplegic spasticity is a measure of the extent of the lesion. About one-sixth of these children die from phthisis.

*Syringomyelia.*—In this affection one or more cavities are found in the spinal cord, most often in the cervical region. The cavity may be merely a dilatation of the central canal, or a space in communication therewith, congenital in origin, lined by the same kind of epithelium, and surrounded by sclerotic tissue. Or there may be excavation, the result of softening and liquefaction of a gliomatous or sarcomatous growth which infiltrates the cord and greatly increases its bulk. The first type is the one which chiefly occurs in children. Bramann (1899) reported cases of symmetrical gangrene of the fingers and toes in 3 brothers, aged 7-13 years; and Bruns (1903) an instance of familial syringomyelia in 4 out of 5 children, which began in each at 17 years of age with perforating ulcers of the balls of the great toes. The symptoms vary with the site and extent of the excavation, and are muscular, sensory and trophic. Other congenital defects of the nervous system may be present.

The intrinsic muscles of the hand waste and the hand becomes claw-like, the shoulder muscles waste and lateral curvature is not uncommon. If the bulb is involved, there may be hemiatrophy of the tongue.



Extension of the affection upward causes nystagmus, ocular palsies, and difficulties in articulation and deglutition. Signs of sclerosis and pressure on the motor tracts may arise, e.g. spasticity. The characteristic sensory change is loss of sensation to heat and cold, usually combined with analgesia. Heat may be felt as cold and cold as heat. Tactile sensation may be normal or completely abolished. These patients are very liable to burn themselves. Excessive redness and sweating, of a limb or generalised, result from vasomotor and secretory disturbance. Arthritic changes of the type of Charcot's joint, perforating ulcer, atrophy of bone, acromegaly or cheiromegaly may arise. *Morvan's Disease*, described by him as occurring only in the upper limb, is a variety characterised by multiple painless whitlows, secondary loss of portions of the extremities, vasomotor symptoms and muscular atrophy. The symptoms of syringomyelia are rare before 15 years of age. If of congenital origin, the disease may remain stationary. It is progressive when due to new growth, and liable to neoplastic formations in the congenital variety. Death is often due to intercurrent disease. Treatment is general and symptomatic.



# CHAPTER L.

## MENINGITIS.

*Purulent—Cerebrospinal; Epidemic and Sporadic—Post-basic—Tuberculous—Syphilitic—Pachymeningitis.*

Meningitis or lepto-meningitis is a general inflammation of the pia-arachnoid. There are probably as many varieties as there are microbes which attack the meninges. All cases are cerebrospinal, but the extent of the spinal inflammation is variable and may be trivial. Careful examination always reveals extension to the brain, so the affection is strictly a meningo-encephalitis. The infective organism reaches the meninges through the blood or lymph, or by direct extension from adjacent structures. Predisposing causes are lowered vitality, injury and heat-stroke. The symptoms vary little with the type, and may be absent throughout or obscured by those of the primary disease. The main features are headache, intracranial pressure, tense fontanelle, psychical changes, cerebral malnutrition and irritation, and generally fever. The age-incidence of the most frequent varieties is shown in the following table, based on a series of 125 cases; males 66, females 59.

*The Age-Incidence of Meningitis.*

	Months 0-6	Months 6-12	Year 1-2	Year 2-3	Year 3-4	Year 4-5	TOTAL	Over 5 Years
Cerebrospinal	15	11	4	3	2	0	35	1
Pneumococcal	5	3	2	0	1	0	11	0
Tuberculous...	3	7	16	13	10	5	54	17
TOTAL ...	23	21	22	16	13	5	100	18

Of these 125 cases 111 were fatal and 88 were examined after death. Of 71 tuberculous cases 58 were verified after death and 54 were under 5 years of age. All the pneumococcal ones were fatal and verified post mortem; the youngest was only 3 days old. Out of 36 sporadic or post-basic cerebrospinal cases 35 were under 4 years of age. The diagnosis was verified



after death in 20 out of 24 which were fatal and by lumbar puncture during life in some of the remainder. Only 5 were ascribed to ear disease and 2 to injury. Other cases ending in recovery have been omitted because of an element of doubt in the diagnosis.

*Classification.*—The division into primary and secondary meningitis, according to the discovery or not of a primary source of infection, is valueless. The division into meningitis of the convexity and that of the base is of little use, for both are frequently involved. The most useful classification is the following :—

(1) **Septic, Suppurative or Purulent.**—This is an acute secondary meningitis due to many organisms and divisible according to its bacteriology into :—

- (a) *Pneumococcal*, secondary to pneumonia or direct infection from the nose ; sometimes primary.
- (b) *Influenzal*, secondary to affections of the naso-pharynx, adjoining sinuses, and otitis media ; often a mixed infection. The organism has been found in the cerebrospinal fluid during life and in the exudate after death.
- (c) *Typhoidal*.
- (d) *Pyogenic*, commonly streptococcal or staphylococcal ; secondary to injury, bone disease, and scarlet fever.
- (e) *B. Coli Infections*.

Purulent meningitis cannot be differentiated absolutely by clinical, pathological or bacterial evidence from the next group, and is sometimes included in one group named “ Acute Meningitis.”

(2) **Cerebrospinal Meningitis.**—This is an acute primary meningitis, due to the diplococcus intracellularis meningitidis or meningococcus, and sometimes to the pneumococcus. It may be epidemic or sporadic in occurrence. Post-basic meningitis is also of this type.

(3) **Tuberculous Meningitis.**

(4) **Syphilitic Meningitis.**

(5) **Pachymeningitis.**

All forms of meningitis are anatomically much alike. Every organism produces more or less the same kind of exudation. In the epidemic type the amount of exudate is considerable and often greater in the membranes of the cord than in those of the brain. It consists of polymorphs in a sero-albuminous fluid. The meningococci are found in the protoplasm of the neutrophilic leucocytes. Lymphocytes and mononuclear cells are present in much smaller numbers. In later stages these cells may predominate over the polymorphs. In pneumococcal meningitis the exudate is much



richer in fibrin and that of streptococcal meningitis is almost the same in character; the polymorphs far outnumber the mononuclear cells. In the tuberculous disease mononuclear cells are predominant. Fibrin is abundant in the exudate of acute cases but in the more chronic forms it is scanty or absent.

**Purulent Meningitis.**—*Syn.: Septic or Suppurative Meningitis—Acute Meningitis or Meningo-encephalitis.*—This variety includes all those acute attacks which are not due to the meningococcus. It is characterised by the effusion of pus or purulent lymph, and generally, but by no means always, affects the convexity of the brain. Its localisation depends mainly upon the primary cause. It may occur at any age, even in the newborn. Of 12 recent cases, 11 were under 4 years of age. The exciting causes were a blow on the head by a cricket ball, purulent pleurisy 2, otitis media 2, and epiphysitis 1; and the other 6 were probably meningococcal. The usual causes are blows on the head, heat-stroke, extension of septic inflammation from the scalp or bones, extension of inflammation from the nose, ear or orbit, rupture of an intracranial abscess, specific fevers, erysipelas, pyæmic conditions, and infective endocarditis, influenza, purulent pleurisy and pneumonia. It is stated that the middle ear is by far the most frequent source of infection; but much too great importance has been attached to otitis media. This is a common terminal affection in children dying from all causes, and in meningitis is quite as likely to be secondary as primary. It is generally stated that young infants are particularly liable to extension of inflammation from the middle ear to the meninges, along the connective tissue passing from the tympanic mucosa and mastoid cells through the petro-squamous fissure to the dura mater. Possibly infection may be carried along the sheaths of arteries, veins or lymphatics. If the inflammation is confined to the tympanum and does not involve the labyrinth there is little danger, but the labyrinth may be affected in infants. On naked eye examination there is often no evidence of direct communication. The otitis media of measles and scarlet fever generally causes sinus thrombosis before it sets up meningitis. The tympanum so often contains sero-pus in infants dying from other diseases, that it seems unlikely to be the source of infection. Were it so, one would expect meningitis to be a much more common disease. Possibly the infective organism gets in through the naso-pharyngeal mucosa or other channels. Extension from the nasal cavity takes place through the cribriform plate of the ethmoid. The disease has followed operative treatment of the naso-pharynx, e.g. removal of tonsils, adenoids and polypi.

**Bacteriology.**—In 25 cases, Netter found the pneumococcus 18, streptococcus 4, meningococcus 2, and other organisms in 3. These results coincide with general experience. The pneumococcus is far the most common organism and may cause epidemics. In cases associated with ear disease, the latter is often pneumococcal. This variety of meningitis may



complicate any inflammatory pleural or lung affection. Primary pneumococcal meningitis has been recorded in a few cases. The next most common organism is the streptococcus, secondary to a general or local infection. The other organisms that have been found are the influenza bacillus, typhoid, paratyphoid, *B. coli*, *B. pyocyaneus*, Friedländer's bacillus, various staphylococci, the gonococcus and even bacillus lactis aërogenes.

*Morbid Anatomy.*—Slight inflammation is limited to hyperæmia of the pia, which is infiltrated with streaks and dots of purulent lymph due to effusion into the subarachnoid space, and therefore adherent. If more pronounced there may be extensive thickening and matting of the membrane, and much lymph and pus. Purulent effusions extend to the subdural space and form a layer on the surface of the brain which can often be peeled or washed off. Pneumococcal cases generally show a thick layer of greenish yellow pus over the convexity in 24-48 hours. It is often most marked on the posterior half of the base of the brain and thus simulates post-basic meningitis. In influenzal meningitis thick greenish pus may be found over the vertex and base, and may extend in a thick layer round the spinal cord, especially posteriorly and laterally. When caused by disease of the nose or ear, the inflammation is most marked at the base and sides of the brain. In other varieties it mainly affects the vertex. The convolutions are flattened by pressure from within. The inflammation affects the choroid plexus and the ependyma so much as to deserve the name of "ependymitis." This produces an increase in the intra-ventricular fluid which is clear, turbid, sero-purulent or purulent. Thrombosis may be present in the cerebral sinuses or the superficial veins of the cortex. In very chronic cases the membranes are thickened and adherent; and the cortex undergoes more or less atrophy and sclerosis.

*Symptoms.*—The onset is gradual or sudden. It is usually sudden, if the child is well previously or if due to rupture of an intracerebral abscess. It is ushered in with headache, vomiting, rigors, delirium and high fever; or with fever, rapid pulse, furred tongue, anorexia, vomiting, restlessness and drowsiness. More frequently the symptoms develop gradually; an irritative stage being followed by coma and paralysis, and preceded by headache, general or localised, and sometimes intermittent. Prodromal signs are those of the antecedent disease, and sometimes may be entirely absent or such as can be ascribed to the disease.

Retraction of the head, rigidity of the neck and opisthotonos may occur quite early, but are much less common than in tuberculous and meningococcal meningitis. Kernig's sign, retraction of the abdomen, and general rigidity may be present. These symptoms depend upon tonic muscular spasm or hypertonia. Convulsions are fairly common and generalised. They may be unilateral, or begin on one side or in one limb and quickly involve the whole body. Muscular twitchings and spasmodic



or continuous rigidity are also common. The fontanelle bulges. Paralysis, usually more or less completely hemiplegic, may be present after the fits, or may arise independently. Convulsions and rapidly increasing coma may be the only symptoms. The mental condition varies. Often the child lies in a condition of stupor or semi-coma; or delirium is present and soon lapses into coma. Teeth-grinding, champing of the jaws and trismus develop in the course of the disease.

Irregular squints and movements of the eyes, nystagmus, conjugate deviation and paralysis of the sixth nerve are often present. The pupils are generally dilated, frequently rigid and unequal. Optic neuritis may be absent throughout or develop in late stages.

The fever is high, 103-105° F.; lowest in the influenzal type. The pulse is frequent, sometimes infrequent, and is not irregular. It becomes abnormally rapid before death. Respiration is frequent or irregular. The reflexes are very variable and alter in the course of the disease. In pneumococcal and streptococcal forms the organism can almost always be recovered by culture from the blood and cerebrospinal fluid.

In the mildest cases the symptoms may be limited to headache, sleeplessness, mental disturbance, slight retraction of the head and moderate fever. These cases are difficult to diagnose from otitis media and from encephalitis. Sometimes the disease is "*latent*," that is it gives rise to no distinctive features, when it occurs as a terminal event in the course of an acute disease, and is only discovered at the autopsy. Consciousness may be present till a few hours before death, or there may be marked somnolence with a tense pulseless fontanelle in infants.

Primary pneumococcal meningitis is usually severe and rapidly fatal, but it may be mild. It can only be diagnosed by lumbar puncture. A boy, 9 months old, developed fever, vomiting, head retraction, and diarrhœa with offensive stools at the onset, followed by constipation. The fontanelle was tense, temperature range 103-105° F., knee jerks active, and Kernig's sign present. The breathing was rapid and the course like that of pneumonia, the temperature falling on the tenth day. The child never seemed very ill and recovered. The pneumococcus was found in the cerebrospinal fluid.

*Course and Prognosis.*—The outlook depends upon the cause and the acuteness of the symptoms. It is best in pneumococcal cases and those secondary to heat-stroke. Under other conditions the prospect is bad. The influenzal type, though purulent, is not always fatal. The duration averages 8 or 9 days, and death is due to convulsions, or cardiac or respiratory failure. Even if recovery takes place it is common for some mental defect to remain or motor defects to ensue.

*Diagnosis* depends upon the conjunction of symptoms of acute meningitis with some cause likely to induce a suppurative form of disease. The child must be carefully examined and the history inquired into for



evidence of one or other of the etiological factors above mentioned. It is chiefly likely to be mistaken for tuberculous or cerebrospinal meningitis, encephalitis, acute otitis media, and enteric fever. If the process is limited to the convexity of the brain, cerebral symptoms may be absent or masked by those of the primary disease. Deafness is generally associated with facial paralysis, whereas in otitis media it occurs independently and may be unilateral. The examination of the fluid obtained by lumbar puncture is essential to accurate diagnosis (p. 618).

The treatment will be considered in connection with other forms of meningitis.

**Cerebrospinal Meningitis.**—*Syn. : Cerebrospinal Fever—Spotted Fever—Malignant Purpuric Fever—Stiff-neck Fever of America—Acute General Lepto-meningitis.*—The epidemic type of cerebrospinal fever should strictly be grouped with the specific fevers, but on account of the preponderance of cerebral symptoms, it is better included with the other forms of meningitis. Sporadic cases and the post-basic meningitis of infants are due to a similar microbial infection, and their course and symptoms are usually typical of a meningitis. It is due to a systemic infection of microbial origin.

The disease was recognised as a distinct entity in Geneva by Vieusseaux in 1805, and it spread in epidemic form in America from 1806 onwards. Since then numerous epidemics have been reported in many parts of the world. A big outbreak occurred in the State of New York in 1904-1905, and a still bigger one in Silesia. Since 1906 outbreaks have occurred in Glasgow, Edinburgh and Leith, Falkirk and Grangemouth, Belfast, Cork and Dublin, various parts of America, Hamburg, Copenhagen, Naples and Gibraltar. This distribution is interesting, for the outbreaks are most of them in relation to seaport centres in connection with each other.

**Bacteriology.**—Micrococci were found in the blood and exudate by Gaucher (1881); in the exudate by Leyden and Ughetti (1883); in the blood, exudate and protoplasm of the cells by Marchiafava and Celli (1884). The organism was cultivated and described by Weichselbaum (1887). He found it in the ventricular fluid in the polynuclear cells and named it the *diplococcus intracellularis meningitidis*. These results have been confirmed by numerous other observers.

The organism occurs in single cocci, pairs, tetrads or groups, and is markedly intra-cellular. It is aërobic and grows best on nutrose ascitic agar or "nasgar," and forms in 24 hours at 37° C. smooth, moist, grey, translucent, circular and oval colonies with regular outline; it will not grow on nasgar below 25° C. Its vitality is low, and it is rapidly killed by drying and sunlight. Microscopically it is indistinguishable from the gonococcus and, like this organism and the micrococcus catarrhalis, it is Gram-negative. It does not clot milk. It produces an acid reaction in dextrose and maltose media. The micrococcus catarrhalis is smaller, and produces no acid



reaction in these media. The organism produces indol but no gas. Its pathogenic virulence is variable, and the poison is an endotoxin.

It was found by lumbar puncture in 82 per cent. of 810 cases in New York in 1904. Elser (1905) isolated it in 109 out of 130 cases. It may be found in the blood and is then a sign of severity. It is not always present in pure culture and may be mixed with pneumococci, staphylococci, streptococci, *B. coli*, *B. typhosus*, the influenza bacillus, the diplococcus crassus and even the tubercle bacillus. It may be crowded out by these other organisms. A Gram-positive coccus, in other respects the same as the meningococcus, has also been found. Thus the bacteriological evidence is strongly in favour of the disease being due to a general blood infection, by the meningococcus, producing local manifestations and meningitis.

*Morbid Anatomy.*—In early cases nothing is found beyond intense hyperæmia of the pia-arachnoid, perhaps localised, acute congestion of the brain and engorgement of the sinuses. Later on the effusion of serum and lymph leads to the brain, sometimes within 24 hours, being covered with greenish-yellow pus. The exudation is serous, fibrinous or purulent. It is chiefly situated over and around the optic commissure, cerebellum, pons, bulb, and along the Sylvian fissure. It may extend up the sides and appear in patches on the cortex, or cover the whole brain. The brain shows bright red spots on section, the “puncta vasculosa,” due to dilatation of the vessels. The cortex is œdematous and shows cell infiltration of the superficial layers. It may be softened locally (focal encephalitis) or generally. The fluid in the ventricles varies in amount, and in character from mere turbidity to pure pus. The cord exhibits similar hyperæmia of the membranes and exudations of variable character, chiefly on the posterior surface. Occasionally the whole cord is covered with thick purulent lymph. The lungs and pleura may show bronchitis, broncho-pneumonia, atelectasis, hypostatic congestion, pleurisy and pleural petechiæ. Petechiæ may be found over the heart and pericardium; and serous, hæmorrhagic or purulent pericarditis may be present. An enlarged spleen is found in about half the cases. The mesenteric glands are enlarged and congested; the solitary follicles enlarged and bright red; and the small intestine engorged. This lymphatism was specially noted by Wettenhöffer in the Silesian epidemic, and is said to follow intraspinal inoculation in monkeys. Other anatomical changes are due to complications, viz., eye and ear disease, purulent arthritis, endocarditis, enteritis and nephritis. Patients, who have recovered and died subsequently from other disease, usually show thickening and adhesion of the pia to the brain, local opacities and thickenings, and occasionally areas of sclerosis and hydrocephalus.

*Mode of Infection.*—There is a certain amount of evidence that the nose is the chief channel of infection, for the organism has been found in the nasal discharge in 25-50 per cent. of the cases, and even in the unaffected. Possibly the infection is conveyed, via the adenoid tissue in the naso-



pharynx, through the lymphatics and blood vessels. On the other hand it is very rare for inflammation of the meninges to develop by continuity from the ethmoid cells, and it is uncommon to find meningitis over the inferior part of the frontal lobe. Perhaps the nasal cavities are infected from the meninges. A possible source of infection is through the conjunctiva, for conjunctivitis is a frequent early sign. Wettenhöffer (1906) regards the tonsils as the probable source of entry, and states that the risk of infection varies directly as the size of the tonsils. The throat is often found injected. There is no reliable evidence that the disease is conveyed by contacts, and meningococci were not found in the throat in such cases in the Belfast epidemic. Another argument against nasal infection is the fact that the lesions in the spinal cord are of older standing than those of the brain. Brain symptoms may be slight, though pus is found in the canal. The early loss of abdominal reflexes also suggests a cord infection. Another view is that it is spread by infected dust, for outbreaks may occur in the same house after long intervals. Possibly it is a form of food infection for it rarely occurs in the breast-fed. It is most frequent among the poor in whom the liability to food infection is great. It is especially common in children, who may acquire the infection through crawling about floors, playing in dusty streets, and putting various objects in their mouths. Further support of the hypothesis of intestinal infection is found in the marked abdominal lesions. The intestines are engorged, and the solitary and mesenteric glands enlarged.

*Etiology.*—Epidemics are due to increased virulence of the organism and an associated depressant factor. Outbreaks are frequent in barracks and under all conditions of overcrowding and bad hygiene. School epidemics are rare, and the disease is not common among the better classes. Lower animals, especially horses, are liable to it, but there is no evidence of the communicability of the disease from animal to man. In the occurrence of house epidemics and other outbreaks it is paralleled by similar outbreaks of pneumonia and typhus. It was probably spread by coughing and sneezing in the few cases of direct infection on record. There is no reliable evidence that it is spread by contacts. Isolated sporadic cases are not infrequent in various districts and at different times of the year, but they do not act as foci for the spread of the disease. It is therefore only mildly contagious. Those outbreaks which occur as a complication or sequel of infectious fever, pneumonia or epidemic diarrhoea, are possibly due to other organisms. The disease is most common in winter and spring, perhaps on account of the liability to nasal catarrh, exposure to cold, and the lack of sunlight.

Both sexes are equally liable, especially in childhood, but in later life it is much more prevalent in males. Children with a tendency to lymphatism, e.g. adenoids and enlarged tonsils, are particularly susceptible. It is most frequent under the age of 5 years; after 10 years it is distinctly less



common, and after 30 decidedly rare. It has been recorded as early as 3 weeks of life. From 70-90 per cent. of the fatal cases are under 15 years of age. In New York 67 per cent. of 2180 cases were under 10 years and of these 15 per cent. were under 1 year.

*Symptoms.*—The incubation period is uncertain, probably from 1-4 days. There are two main types of the disease :—(1) Toxæmic, malignant or fulminating, with profound blood poisoning; and (2) Meningitic. The onset is sudden in about 90 per cent. It may be extremely gradual, no symptoms being noted before hydrocephalus has begun. For a few hours or days there may be premonitory symptoms such as headache, languor, anorexia, chilliness, pain in the back and limbs, stiffness of the neck muscles, a full and strong pulse, and temperature 101-102° F. In an acute toxæmic sporadic case in an infant, 18 months old, the first symptom was pain in the ankle of a few hours duration; this was followed by convulsions for 24 hours, temperature 105° F., dyspnœa, very frequent pulse, unconsciousness and other signs of a profound toxæmia, suggestive of a pneumococcal infection. Definite meningitic symptoms developed in a few days. The usual onset is in the latter half of the day with vomiting, stiffness of the neck or retraction of the head, severe occipital headache, high fever, rigors or chilliness and convulsions. Headache is less marked in children than in adults and convulsions are more common. The child rapidly passes into a state of stupor or delirium; and coma may come on in a few hours. In the toxæmic, malignant or fulminating cases are found great depression, muscular spasms and purpuric rash, with moderate fever and perhaps a pulse rate as low as 50. Thus a 4-month old, well-nourished baby had a little abdominal pain and in a few hours was distinctly drowsy; a few bright red purpuric spots were found; the pulse rate and respirations became very frequent, pupils small and unequal, and death occurred within 36 hours, preceded by a discharge of turbid fluid from the nose, mouth and left ear. Many more spots had come out on the upper part of the back, and large patches of purplish discoloration on the back and thighs. Pain in the limbs and trunk are often present, sometimes temporarily; and giddiness or vertigo is not uncommon. Inflammatory conditions of the tonsils, fauces, pharynx and posterior nares have been noted at the onset in many acute cases, especially in the Silesian epidemic.

In a typical case the child lies on the side with the head retracted, spine arched forward and the limbs flexed; the neck muscles are very rigid and resist forward flexion of the head. It is restless, screams from pain, and exhibits general hyperæsthesia, sometimes tenderness over the cervical vertebræ. The headache may be referred to any part of the skull and is severe. Pain continues during the acute stages and, besides being felt in the head, is also referred to the back of the neck and spine and almost any part of the body; the slightest movement may cause most severe pain. It usually diminishes during the second week.



Muscular contractures result from the irritation and give rise to the stiffness of the muscles of the neck and back, retraction of the head, opisthotonos and flexion of the limbs. It comes on in the course of 2 or 3 days, may persist for some weeks, and disappears slowly. The rigidity of the neck may vary from day to day and is usually the last sign to disappear. Teeth grinding, twitching of the mouth angles, twitching of the hands and arms, rolling of the eyes and squint are frequently present.

Hyperæsthesia is more marked in this than in any other form of meningitis. It may even be detected when the child is almost comatose. There is marked sensitiveness to cold. The skin may exhibit the *tâche cérébrale*. In bad cases a dusky mottling is seen during the first 2 weeks, and in the worst the skin is covered with subcutaneous hæmorrhages, varying from minute red dots to bluish spots and larger patches like bruises. This purpuric eruption gave rise to the name of "spotted fever," but it is quite uncommon nowadays. The rash may come out in crops and is sometimes like that of typhoid. Towards the end in fatal cases are found fine punctate hæmorrhages over the abdomen, and even larger subcutaneous bruises are not uncommon. A rash occurs at some period in about 20 per cent. Herpetic eruptions about the lips and angles of the mouth, and occasionally on the other parts supplied by branches of the fifth nerve, occur in about 10 per cent. Erythematous and roseolous rashes, suggestive of scarlet fever or measles, have also been noted.

Reflex action is often abolished early. The knee jerks are sometimes exaggerated at the onset, and almost always absent later; occasionally they are variable. The abdominal reflex is lost early. There is no ankleclonus and no Babinski sign. Kernig's sign is more often present than absent. It is frequently difficult to elicit under the age of 2, and usually present after that age.

The tongue is moist and slightly furred; appetite impaired or lost; and thirst and constipation may be present. Swallowing may be difficult because of the retraction of the head or paralysis of the muscles of deglutition. There is no special tendency to constipation, and diarrhœa is by no means an infrequent complication. Retraction of the abdomen is not a notable symptom. Vomiting is common at the onset and during the course of the disease, reappearing with exacerbations of the morbid process. It is sometimes frequent in protracted cases and relieved by lumbar puncture. It is of the cerebral type, unaccompanied by nausea, and sometimes explosive or projectile. In later stages it is due to hydrocephalus. Loss of flesh and strength is rapid and severe, much more so than in tuberculous meningitis. The spleen is sometimes enlarged and the glands are often hard and shotty.

The pulse is increased in frequency in proportion to the severity of the attack, but it may be slow and fall in severe cases. It becomes less frequent and irregular from coma due to intra-ventricular effusion. It often varies



greatly in frequency at different periods of the day, but it rarely presents the intermissions and characteristic irregularity of tuberculous meningitis. The ratio of pulse and respiration is irregular. Towards the end the pulse becomes uncountable. Marked leucocytosis, with diminution or absence of eosinophiles, is found on examination of the blood in bad cases. Myocarditis was noted as common and a frequent cause of death in the Lisbon epidemic.

The respiration is increased in frequency, occasionally sighing or irregular, sometimes of the Cheyne-Stokes type. Cyanosis may be present in malignant cases apart from any cause in the heart or lungs.

The temperature is variable, generally proportionate in height to the severity of the attack, but it may be low or subnormal in bad cases. As a rule it rises to 103°-105° F. and becomes hyperpyrexial towards the end. In malignant cases hyperpyrexia may be present at the onset. Great variations in the height of the fever may be noted in the course of a few hours. The usual range is 100-105° F., and it is often higher in the morning than the evening. Sometimes the fever is of the continued type at the onset and after that markedly hectic. Its duration varies. Frequently it lasts 4-6 weeks, and often much longer.

The urine is normal in amount or increased. Blood and casts have been found in it; but hæmorrhagic nephritis and paralysis of the bladder are rare complications. Retention is apt to be overlooked. Incontinence of urine and fæces is a frequent symptom.

The mental condition shows every grade of disturbance from simple apathy to coma or acute delirium. Usually the initial irritability and resentment of disturbance are quickly followed by stupor and coma. Except when comatose the child is restless from pain and delirium. If the delirium in early stages is violent, it is short and soon passes into coma. Taste and smell are difficult to test and are rarely affected. Otitis media or interna may cause permanent deafness, but such deafness can arise quite independently from cerebral causes and prove permanent.

The eyes show a great variety of changes. At the onset they are suffused from slight conjunctivitis; photophobia and squint are common, and the pupils small. Conjunctivitis is often present and may be intense. Retraction of the eyelids may be noted as early as the fourth day, but is more frequent in chronic cases. The motor symptoms include palsy of the third, fourth and sixth nerves giving rise to ptosis, squint and diplopia. External squint is frequent and variable during the acute stages. It is commonly due to nerve irritation and rarely permanent from nerve palsy. Conjugate deviation is caused by cortical inflammation or lesion of the nucleus of the sixth nerve. Paralysis of the ophthalmic division of the fifth nerve and anæsthesia of the cornea are probably antecedent causes of corneal ulcer. In later stages the pupils are dilated, sometimes unequal, and in bad cases respond feebly to light. Optic neuritis is infrequent and



late in development, and may be followed by atrophy. Blindness may be present from no obvious cause and possibly due to affection of the occipital cortex. A plastic and suppurative choroiditis, with iritis and occasionally pus in the anterior chamber, may occur early or late in the illness. It is sometimes called panophthalmitis, metastatic choroiditis, or pseudoglioma. In one acute case it commenced in the first week and in another chronic case it was treated in the eye department for some 2 or 3 weeks before meningitis was suspected. Panophthalmitis is rare.

Convulsions, local or general, may occur at the onset or termination and less often in the course of the illness. Other complications are hydrocephalus, aphasia, hemiplegia, paralysis of the muscles of deglutition, and choreiform movements; acute multiple arthritis; pneumonia, bronchopneumonia and pericarditis; and acute catarrh of the nasal fossæ and accessory sinuses in the nose.

*Varieties.*—An ambulatory type is very rare. Abortive attacks are common in some epidemics. The onset is severe but the symptoms subside in a day or two, and convalescence is rapid and complete. An intermittent type is characterised by alternate irregular intermissions. Chronic cases last for months with recurrences. On grounds of severity they can be divided into acute fulminant, ordinary, mild, abortive and chronic cases.

Isolated or sporadic cases differ little from those in epidemics. As a rule they are milder in type and gradual in onset, though sometimes ushered in with vomiting or fits. The meningitic symptoms may be well marked or limited to headache, vomiting, retraction of the head, pain in the neck, and fever. Rashes are infrequent but herpetic eruptions are more common. It is almost impossible to diagnose the disease from suppurative meningitis due to other causes, except by bacteriological examination of the fluid obtained by lumbar puncture.

**Post-Basic Meningitis.**—*Syn. : The Cervical Opisthotonos of Infants—Simple Post-basal Meningitis—Posterior-basal Meningitis—Chronic Basilar Meningitis—Occlusive Lepto-meningitis.*—This disease is called “posterior basal” because it affects chiefly the posterior fossa of the skull, the inflammatory products accumulating in the basal arachnoid systems of Key and Retzius. Clinically it is a variety of cerebrospinal meningitis, often very chronic, and may end in recovery.

*Bacteriology.*—A diplococcus was found by Still in 7 out of 8 cases, and by Thursfield in 8 out of 9, no other organism being present. It is probably identical with that of Weichselbaum, merely differing in virulence. It is aërobic, grows well on agar and in broth at 37° C., but does not grow at 20° C.; does not grow on potato or curdle milk. It is Gram-negative, closely resembles the gonococcus, and as a rule is smaller than the pneumococcus. It presents no lanceolate forms, and its opposed surfaces are more or less flattened and not always equal. It grows more freely in broth and has greater vitality than Weichselbaum’s organism. Nevertheless Jaeger of



Stuttgart has kept the latter organism alive in broth for 14-17 days; and Netter (1903) states that he has kept it alive for more than 4 years by subculture in broth.

Agglutination tests seem to indicate a difference between these organisms. In the Belfast epidemic the blood agglutinated its own meningococci, but not those of simple basal cases; and the blood of basal cases reacted to cocci of their own type but not to those of the epidemic type. The organism is seldom present in great numbers. It may be associated with the pneumococcus, staphylococcus, and the influenza or tubercle bacillus, but is usually found in pure culture. It has not been found in the blood. Cultures should be made from the sub-pial exudation at the base of the brain, the intra-ventricular fluid or that obtained by lumbar puncture.

*Morbid Anatomy.*—The inflammation is limited to the base of the brain, chiefly the interpeduncular space and the region between the bulb and cerebellum, the ventricles and the upper part of the cord. The under surface of the cerebellum and the apices of the temporo-sphenoidal lobes are often involved. It may extend all over the base of the brain, right down the cord, and rarely on to the vertex. The pia-arachnoid is thickened and injected, adherent and covered with greenish-yellow lymph. Usually the inflammation begins over the fourth ventricle. The intra-ventricular and sub-arachnoid fluid is increased, turbid, and contains flakes of lymph. The foramina of Majendie and Luschka may be blocked. The viscera are unaffected. Peri-arthritis has been noted, with the exudation of thick adhesive lymph round the tendon sheaths of the joints and even pus in the joints, the specific organism being isolated therefrom.

*Etiology.*—Heredity is unimportant and there is no special sex incidence. The disease is common under 1 year of age, and rare after the second year (p. 642). From 50-75 per cent. occur in the first year of life. In the epidemic disease 50 per cent. are under 10 years. The seasonal prevalence varies, but it is always prevalent though not common. The cases are more or less equally distributed throughout the year. Sometimes it is more frequent in hot weather, at other times when it is cold. Occasionally the number and distribution of the cases suggest an epidemic character.

The predisposing causes are catarrhal affections, infective disorders, general debility, otorrhœa, injury, and alimentary infection. Yet it occurs in the strong, breast-fed and healthy. The little evidence there is of the channel of infection is in favour of spread from the naso-pharynx via the Eustachian tube, middle ear, and petro-squamosal suture. Catarrh of the naso-pharynx is a frequent antecedent. Otitis media is uncommon, and if present may be secondary.

*Symptoms.*—The onset is usually sudden with vomiting, convulsions or head retraction, bulging fontanelle, screaming and fever. These symptoms are more or less associated or follow each other quickly. Mild cases



may start quite insidiously, and attract little attention until there is evidence of hydrocephalus. In others there are irregular fever, headache, vomiting, head retraction, bulging fontanelle, somnolence and rapid pulse. Languor and irritability quickly supervene on an acute onset. The intellect is dull, and the child lies quiet and unobservant. Drowsiness, coma and delirium are occasionally present in acute cases, or as the result of hydrocephalus.

Retraction of the head is a cardinal sign in young infants but may be absent in older children, though usually rigidity of the neck muscles is present. It is probably the position in which the meninges are most relaxed and kept at rest. It may be associated with opisthotonos, coming on spasmodically and so severely that the occiput and sacrum or heels are approximated, or the child rests on the head and heels. In its extreme form it is rarely persistent but it never quite disappears, and a certain amount of rigidity of the neck muscles and retraction of the head is always present. Later, the head may fall back on account of paresis of the neck muscles. Retraction of the head, vomiting, wasting, and a variable degree of fever may be the only signs present throughout. The retraction is commonly associated with general tonic rigidity of the limbs, most marked in the legs, which are fully extended, often crossed, with the feet arched and the toes pointed or flexed. The arms are extended, adducted and overpronated. This rigidity may be replaced by intermissions of flaccidity. Tonic spasms may alternate with clonic ones, and general convulsions are not uncommon though less so than in other types of meningitis. The knee jerks are exaggerated. A mask-like rigidity of the face, retraction of the angles of the mouth, teeth grinding, champing movements of the tongue and jaws, and sucking movements of the lips are sometimes noted.

Vomiting is frequent and explosive. It may be the most marked symptom, or slight and comparatively unimportant. Anorexia is common; and severe head retraction may interfere with swallowing. Emaciation is great and rapid, especially if there is much vomiting or enteric disturbance. Constipation is rare, and enteric catarrh or simple diarrhoea not infrequent. The abdomen presents no special feature unless there is flatulent distension from enteric trouble, or retraction from emptiness due to continued vomiting. The *tâche cérébrale* is frequently absent.

Ocular symptoms are often absent or present in variable degrees. Retraction of the upper lids is common. The eyes are wide open and staring; a marked contrast to the photophobia and closed eyes of tuberculous meningitis. Squint is variable and transient, ocular palsy rare, and nystagmus, pseudo-nystagmus or an oscillation of the eyes common. These signs are due to the spasmodic rigidity. The pupils are usually normal; may be unequal, dilated, and oscillatory; or pin-point in early stages and dilate as hydrocephalus develops. A transient amaurosis, the "*fleeting amaurosis*" of S. Stephenson or "*acute cerebral amaurosis of*



*infancy*” of Gay, has been described. It occurs in about one-third of the cases, or even more frequently, being present at some period of the illness, sometimes not until 1 month after the onset. It begins insidiously, occasionally suddenly. It lasts for 3-6 or more weeks, even for years, and probably is never permanent. Vision returns gradually. It is not due to hydrocephalus, for it may disappear though the latter progresses. The pupils are slightly enlarged and react little if at all to light. There are no fundus changes. It is due to inhibition of lower visual centres in the brain. Optic neuritis is relatively rare, occurring in perhaps 15-20 per cent. Primary atrophy may result from hydrocephalus. Pseudo-glioma is an occasional complication.

Labyrinthitis may occur early in severe cases, but is more common in mild ones. It is often associated with otitis media and is generally bilateral. In older children it gives rise to giddiness.

The head does not alter unless hydrocephalus develops. The fontanelle bulges, and the superficial veins of the scalp are prominent. Later, the sutures may separate. The circumference of the head must be measured weekly.

The pulse rate is increased. Leucocytosis is considerable in early stages. Vasomotor disturbance is uncommon. The respiration may be accelerated. Breathing is sometimes sighing, and yawning may be present. In late stages “cyclic” or “cerebral grouped breathing,” a pause following groups of 3-6 respirations of equal depth, is present but is of no prognostic value. True Cheyne-Stokes breathing is rare and a bad sign.

The temperature is most variable. As a rule it is of the irregular hectic type. Sometimes there is little or no fever; or a continuous high fever at the onset; or periodical large oscillations of a hectic type and at varying times of the day. The temperature may rise 6° or 7° in a few hours, and fall again in another few hours or not for half a day. Such paroxysms may occur daily, or at irregular intervals, and are generally accompanied by headache, chilliness, and increase in the symptoms.

*Course and Prognosis.*—Gradually the child loses flesh and strength, develops hydrocephalus, and dies from asthenia, marasmus or terminal convulsions. In a few cases the cerebrospinal fluid has been discharged through the nose. Erythematous rashes are not infrequent. Arthritis is a rare complication. Recovery may be complete or various sequelæ may be left, notably hydrocephalus, deaf-mutism, various degrees of imbecility and general rigidity. The prognosis improves with the age of the child. Few under 1 year recover, and few over 3 years die. The mortality of cases admitted into hospital is about 70 per cent. Probably many mild cases are not diagnosed and the real mortality is not over 50 per cent. Relapses and intermissions are frequent. The average duration of fatal cases is 6-8 weeks, but the disease may prove fatal in 1 week or may last for 3 months.



Some infants die subsequently from hydrocephalus, and even quite suddenly after apparently perfect recovery.

The *diagnosis* depends on the age of the child, the marked retraction of the head, rigidity, retraction of the upper eyelids, amaurosis without fundus changes, the absence of constipation, the slight degree of psychic disturbance, and the prolonged indefinite course. It cannot always be diagnosed from the tuberculous variety in early life, except on careful examination of the fluid obtained by lumbar puncture. Sometimes it is difficult to differentiate the retraction from the reflex opisthotonos due to otitis media, hydrocephalus, teething, gastro-intestinal causes, cerebral tumour, typhoid fever, and chronic tetanus.

**Diagnosis of Meningitis.**—Cerebral symptoms are so common in acute febrile diseases, especially lobar pneumonia, that meningitis is frequently diagnosed when it does not exist. Cases of sudden onset, with vomiting and high fever, simulate scarlet fever. Sporadic cases and those of mild type are apt to be ascribed to digestive disturbances or teething. It is often difficult to exclude reflex causes of head retraction. Internal hydrocephalus may be the only sign, and it is impossible, in the absence of a definite history of an acute onset, to decide whether it is of primary origin or secondary to basal meningitis. The epidemic is almost identical with the suppurative form. In the former a sudden onset, marked hyperæsthesia and pain, and retraction of the head, are more likely to be present; in the latter the history of a cause, or the presence of a primary affection likely to give rise to it, is often of assistance. Tuberculous meningitis is generally of more gradual onset with less fever and prostration, less pain and general hyperæsthesia. The examination of the cerebrospinal fluid yields valuable evidence. The epidemic type may have to be diagnosed from typhus fever, malignant exanthems, influenza, cerebral pneumonia, sunstroke, the hyperpyrexia of acute rheumatism, gastro-enteritis, and broncho-pneumonia. Outbreaks due to the pneumococcus can only be distinguished from those due to the meningococcus by bacteriology. Meningococci may not be found at first and are sometimes not present until the twentieth day. The serum does not develop agglutinating properties for some days; a fresh strain of cocci must be employed.

**Prognosis.**—Pneumococcal and streptococcal cases are almost always fatal. The mortality in the meningococcal epidemics is varied and may reach 92 per cent. In some mild epidemics it is extremely small. As a rule about half the cases die; and the mortality is higher at the beginning than at the end of an epidemic. The initial convulsions may prove fatal. In the New York epidemic 7 per cent. died on the first day; 34 per cent. in the first 5 days. Fatal cases usually terminate in a week. The milder sporadic cases and post-basic ones in infants are often very protracted. An acute sporadic case in a 4-month old, breast-fed, fat child ended fatally within 36 hours. Another one, even more severe at the onset, ended in



recovery after several weeks of severe illness. In acute attacks the prospect of recovery is increased if the first fortnight elapses without any serious complication. Convulsions, high fever, delirium or coma, dyspnœa and an unduly frequent pulse are unfavourable signs.

Bad symptoms may subside and the child seem well on the road to recovery when a relapse or recrudescence occurs and ends fatally. Relapses usually last 3-4 days and occur at intervals of 4-8 days. The illness may be prolonged even for months and yet the child may make a perfect recovery. Lung complications, especially in infants, increase the gravity. Subcutaneous hæmorrhages and nephritis indicate severe blood infection with probably a fatal issue. Death may be due to the disease, complications or sequels.

The prognosis as regards the ultimate effect on the nervous system must be guarded. Mental impairment, from mild forms of irritability, temper and headache, up to complete imbecility and all grades of mental disturbance and insanity, may ensue. Other sequels are peculiarities of temper, morals and emotions; deafness or blindness; convulsions and epilepsy; nystagmus and ataxia; monoplegia or hemiplegia; secondary contractures, temporary or permanent; and hydrocephalus which may be stationary or progressive.

**Treatment.**—Prophylaxis is of little importance, beyond the general measures for the maintenance of health. In epidemics the use of the nasal douche and formamint lozenges, the isolation of “contacts” and “intermediaries,” and the thorough disinfection of houses and the destruction of possibly infected food should be adopted.

In the early stages an attempt must be made to reduce the cerebral hyperæmia by cold or ice to the head and nape of the neck, and in very bad attacks to the dorsal and lumbar spine, keeping the extremities warm. Keep the room dark and cool. Quiet is essential, the patient must not even be moved from one room to another. The eyes may be bandaged and the ears stuffed with cotton wool. Opium relieves restlessness, pain and irritability. Hydrotherapeutic measures include “steaming,” hot packs or baths for 15-20 minutes twice a day, with ice bag to the head. They relieve headache and irritability, promote sleep, reduce delirium and the tendency to convulsions, lessen rigidity and allay rigors. Tepid sponging is sufficient in milder cases. The cold pack may be needed to reduce high fever. Counter-irritants to the spine are of doubtful advantage. Bleeding is apt to prove injurious. The local application of 1-3 leeches to the temples or mastoids may possibly relieve headache and prevent coma or convulsions in children.

The chief hope consists in maintaining the strength by careful nursing and judicious feeding. The diet should be simple, nutritious and easily digested. Nasal feeding may be required. The child should be fed as well



as possible without upsetting its digestion. Wasting is often extreme and gain in weight is a favourable sign.

Drugs are of little use except for the relief of symptoms. Bromides are given to diminish the irritability of the brain and cord, and promote rest. For headache apply ice locally and give bromide, phenacetin, phenazone, chloral, codeia, morphia, and hot baths. For severe vomiting order cracked ice, ice to the epigastrium, temporary starvation, careful feeding on small amounts of peptonised milk, bismuth, dilute hydrochloric acid before food, lumbar puncture and morphia *sub cutem*. Calomel or saline cathartics should be given for constipation; chloral and bromides for pain and restlessness; caffeine for pulmonary oedema and respiratory paralysis; stimulants for collapse and a typhoid state; morphia injection for severe pain; and for convulsions, chloral by mouth or rectum, opium, chloroform inhalations, morphia *sub cutem*, lumbar puncture and hot baths. Ergot, quinine in large doses, calcium sulphide, pot. iod., and inunctions of mercury and iodoform have been recommended in the early stages of an acute attack. Strychnia should be avoided throughout; so too alcohol during acute stages, unless absolutely essential; and the coal tar derivatives are rarely advisable because of their depressant action.

Lumbar puncture and the removal of fluid reduces intracranial pressure and withdraws toxic products. It can be repeated every 2-4 days, if the fontanelle is tense and the signs of pressure marked. In the Lisbon epidemic the removal of fluid was followed by injections of 9-12 c.c. of 1 per cent. solution of oxycyanide of mercury.

Anti-diphtheritic serum kills pure cultures of the meningococcus but has not proved of value. Of the various serums in the market, namely those made by Kölle and Wassermann, Jochmann, Ruppel, Dopter, and Flexner and Jobling, the last two appear the most useful. A lumbar puncture is done and some of the cerebrospinal fluid permitted to escape. If it is very thick, normal saline fluid is injected and allowed to flow away. After that 30 c.c. of the serum is injected into the canal. The signs of improvement are a fall in temperature, diminished leucocytosis, less severe nervous symptoms, perhaps return of consciousness, and fewer and weaker cocci. The injection is repeated in 12 hours, if there is no improvement, and daily until the temperature falls. Good results may follow the injection of serum although no cerebrospinal fluid is first removed.

Normal serum possesses distinct bactericidal power. Repeated lumbar puncture and withdrawal of fluid is of value, for it causes the exudation of fresh antitoxic fluid to replace the worn-out fluid removed. The organism grows well in cerebrospinal fluid and its virulence is due to endotoxins liberated after its death. It is therefore important to remove the fluid containing the toxin, replacing it by fresh fluid secreted by the patient or by the injection of an anti-serum which, in order to be beneficial, should be used at the onset. Vaccines prepared from the patient's cocci have been



used and followed by recovery but the treatment is still on its trial. Redmann injected the patient's cerebrospinal fluid, obtained by lumbar puncture, subcutaneously in two cases. In one the fever subsided in 3 days and the other also recovered. It is extremely difficult to estimate the value of such lines of treatment. Meningococcal cases vary greatly in severity and even the most virulent ones will sometimes recover quite rapidly under the simplest measures.

**Tuberculous Meningitis.**—By far the most frequent variety of meningitis in children is the basal meningitis due to the tubercle bacillus. Out of a series of 125 cases under 10 years of age, 71 were diagnosed as tuberculous and the diagnosis was verified in 58 after death. Strictly speaking the disease is rarely primary or localised. It is either secondary to tuberculosis elsewhere or part of a general infection. In children the meningeal symptoms may be the only ones present and a positive diagnosis of meningitis can be made, whereas the presence of a local focus or a general tuberculosis can only be inferred. It is, however, extremely rare to find no local evidence of disease elsewhere after death.

Cases may be grouped into 3 classes. In the first are those rare instances in which the disease is limited to the brain and no local focus can be found. Out of 58 post mortem examinations 5, ranging from 2-9 years of age, were of this nature. One of them showed some caries of the ethmoid but it was doubtful whether this was tuberculous. Possibly in every case of primary tuberculous meningitis there is some local focus, e.g. in the ears or naso-pharynx, overlooked at the autopsy. In the second group are those cases in which the child dies from meningitis secondary to a localised tuberculosis with little dissemination. The primary focus is generally a tuberculous gland in the mediastinum or mesentery, and occasionally tuberculous disease of skin, bones, joints, lungs, intestines, nose, middle ear, etc. In the third group are all those cases in which the meningeal affection is part of a general tuberculosis and bears a comparatively unimportant relationship to the disease.

*Etiology.*—The general etiology is the same as that of tuberculosis elsewhere. The age-incidence is of considerable importance. Out of 58 cases verified post mortem, 46 occurred during the first 5 years of life and 12 in the second 5 years. Including other cases not verified after death, 54 out of 71 were under 5 years of age. This may indicate that a child susceptible to tuberculosis usually develops it during the early years of life. Ten proved cases were under 1 year and, of these, 3 were under 6 months, the youngest being 3 months of age. There were 16 in the second, 13 in the third, 10 in the fourth and 6 in the fifth year of life. Of 26 cases under 2 years of age, 16 were 12-18 months old. Thus it is far from rare in infancy.

There is no special preponderance of either sex, possibly a few more cases occur in boys, e.g. 38 boys to 33 girls. The seasonal incidence of 66 cases showed that 19 began in June and July. In 22 cases there was no



tuberculous family history. Isolated cases occur in many families without any tuberculous heredity. On the other hand many cases of tuberculosis in the first year of life are strongly suggestive of direct infection from a tuberculous relative or nurse. Reich (1878) reported 10 fatal cases within 14 months in infants who all had the same phthisical midwife and were children of healthy parentage. Probably they were infected at or shortly after birth. Seven of the 10 died in the fourth month of life.

Injury is a doubtful cause. In 1 patient aged 2 years, who had had a fall 3 weeks before the illness, both her father and sister were "consumptive" and the presence of pleuritic adhesions at the apex of one lung suggested former tuberculosis. The possibility of injury being an exciting cause is of great importance in the case of school children. If the child receives a box on the ear, or a blow on the head, at the hands of the teacher and subsequently dies from tuberculous meningitis, the unfortunate teacher may be hauled up before the Coroner's Court and censured by a typical British jury. The common explanation is that the child is punished for stupidity or inattention, the state of mind so likely to be present during the early stages of the disease. Careful examination may reveal a primary focus. On the other hand it is possible that injury to the brain or meninges may cause local congestion or damage of such a nature as to render the affected parts a suitable soil for the growth of the bacillus, which under less advantageous conditions might have perished. An interval of 2-3 weeks must necessarily elapse between the blow and onset of symptoms to allow for the development of the organism.

Infection by food is incredible apart from the intervention of a local tuberculous process. The development of the disease in young infants and children who have always appeared healthy and have not, as far as is known, been exposed to infection, can be explained by the wide dissemination of the tubercle bacillus and the latency of chronic tuberculous processes. In many instances there is a definite history of a lung affection liable to produce congestion and hyperplasia of the mediastinal glands, rendering them particularly susceptible to tuberculous infection or stimulating a latent focus into activity. Measles and whooping cough have a very evil reputation in this respect. Or there may be definite evidence of active or quiescent tuberculosis in some part of the body. Occasionally dissemination is set up by interference with tuberculous disease of glands, bones or joints by bonesetters or surgeons.

*Morbid Anatomy.*—The anatomical changes may be summed up as basal meningitis with tubercles, granular ependymitis, hydrocephalus, and nutritional disturbance of the brain. The inflammation of the pia-arachnoid, mainly at the base of the brain, is set up by the deposition of tubercle bacilli and the formation of miliary tubercles. At first the tubercles may be unassociated with inflammation. They are small, greyish white or translucent granules, situated along the blood vessels of the pia mater and the



arteries extending into the brain, especially the branches of the Sylvian artery. Gee showed that by washing away the brain substance the tubercles could be demonstrated along the whole length of the cerebral arteries. The bacilli spread by the blood or the lymphatics in the vessel walls.

Even the tubercles may be absent, and the meningitic symptoms due to bacillary infiltration or to toxins. Microscopically such a case shows diffuse leucocytic infiltration of the meninges and superficial brain substance; the cells being in some places aggregated into little nodules. The affection is predominantly perivascular, and a large number of tubercle bacilli are found wherever there is this infiltration, especially in the neighbourhood of vessels.

The amount of apparent disease present in some cases after death is marvellously small, although the symptoms have been well marked. It may be limited to thickening and matting together of the membranes at the base of the brain, chiefly in the interpeduncular space, and a few scattered tubercles. Sometimes the membranes at the base are remarkably thick and opaque or gelatinous, obscuring the underlying structures. Or there may be more or less fibrino-purulent exudation and yellowish or greenish yellow lymph. Purulent exudations are possibly due to mixed infections, and are never so great or extensive as those seen in non-tuberculous meningitis. The inflammatory products are most marked at the base over the optic chiasma, interpeduncular space, pons, and between the medulla and cerebellum. Frequently they extend up the Sylvian fissures, or may be limited to this region, and occasionally extend on to the surface of the brain. Tubercles may be found in patches on the convexity of the brain, between the two hemispheres, and on the folds of the choroid plexus.

The amount of fluid in the pia-arachnoid space varies from normal up to 4 oz. It is usually clear, or slightly turbid from flakes of lymph, and exceptionally is sero-purulent. The brain becomes intensely congested from extension of inflammation and a true meningo-encephalitis results. This leads to thrombosis of small arteries, veins and cerebral sinuses, deficient blood supply, increased venous pressure, effusions, cell degeneration, softening and necrosis. Even in acute cases the brain substance may remain firm. More often it is œdematous, especially at the base, and is universally softened or almost diffuent. The softening may be marked, although there is no excess of intra-ventricular fluid. It is due to impaired nutrition in consequence of circulatory and lymphatic obstruction, rather than to hydrocephalus.

The amount of effusion into the ventricles varies. As a rule it is greater if the illness is prolonged. It is partly dependent on obstruction at the foramen of Majendie and partly on ependymitis, for the ependyma is often inflamed, thickened, red and granular. The fluid is clear, or slightly turbid from particles of lymph and pus.



If the ventricles are much distended the convolutions are flattened. This flattening and an excess of fluid are the first points noted on opening the skull. If the sutures are un-united they may separate and thus modify the effects of pressure. The superior longitudinal sinus is often more or less thrombosed; the cerebral sinuses much distended with dark fluid blood; the superficial cerebral veins similarly distended; and the venæ Galeni blocked by matting of the velum interpositum.

Section of the brain shows softening, puncta hæmorrhagica, and perhaps one or more scattered tuberculous nodules, varying in size from a minute pea to a good sized tumour, situated in any part of the brain and sometimes attached to the meninges. The cortical brain cells are globular or swollen, the nuclei contracted and the protoplasm "dusty" in appearance.

Sometimes the inflammatory process and a few tubercles extend down the spinal cord. Caseating meningitis is rare. It causes symptoms like Jacksonian epilepsy and is of rather longer duration than ordinary cases. A diffuse caseation of the meninges, principally of the vertex along the longitudinal fissure, and a greenish deposit extending into the sulci are found after death.

In 23 out of 27 cases the mediastinal glands were tuberculous; in 2 the lungs were very tuberculous but the state of the glands was not noted; in the remaining 2 the only anatomical changes outside the cranium were caries of the ethmoid in 1 and localised apical pleurisy in the other. The gland generally affected, often the only one, was that situated at the bifurcation of the trachea and sometimes called the "pretracheal" gland. In 4 of the 23 cases the mesenteric glands were also tuberculous. In one of these the peritoneal surface of the diaphragm was covered with miliary tubercles, but there was no macroscopic evidence of the disease in the lungs. In another there were tuberculous nodules in the skin and brain, and general lung dissemination, especially at the right apex. In the remaining two, in which both sets of glands were caseous, one had tuberculous vomicæ in both lungs and two small ulcers in the intestines, and the other had an old caseating and calcifying nodule in the lung. No less than 12 cases showed extensive pulmonary disease of long duration.

*Pathology.*—As a result of predisposing conditions due to inflammatory affections of the lung, or from direct infection, one or more of the mediastinal glands becomes congested, infected with the tubercle bacillus, and undergoes caseation. Such a gland may remain quiescent, calcify, or break down by fatty necrosis. On softening, the tuberculous infection is carried by the blood vessels throughout the body and sets up general miliary tuberculosis. The result may be a meningitis to which the patient succumbs without the bacilli having acquired much of a hold elsewhere. Occasionally there is general miliary tuberculosis and the pia is unaffected. In other instances infection may be carried from tuberculous glands elsewhere or from tuberculous disease in any organ without the intervention of breaking down



glands. The disease is spread through the circulation, rather than by extension or through the lymphatics.

The primary effect on the brain is the formation of minute tubercles in the walls of the smallest arteries. An inflammatory process is set up, inducing an obliterative peri-arteritis and arteritis, or obstruction to the circulation of blood and lymph by inflammatory products at the base of the brain. The nutrition of the brain is interfered with and softening results. Ventricular effusion is partly mechanical and partly due to inflammation of the choroid plexuses and ependyma.

Death ensues from the nutritional disturbance rather than from the actual mechanical effects of retained or effused intra-ventricular fluid. In only about two-thirds of the cases, which I have examined post mortem, was there an excess of fluid in the ventricles and sometimes the quantity was little increased. Many children died comatose although there was no evidence of increased intracranial pressure after death. Nor does the amount of softening depend upon the amount of fluid in the ventricles. It may be marked in the absence of excess of fluid, but as a rule the greater the amount of fluid the greater is the softening. Both may depend upon the same cause.

*Symptoms.*—The onset is rapid in cases of prolonged tuberculosis; gradual and insidious when secondary to latent local disease. In the latter class of case it is usual to divide the course of the disease into:—(1) a *prodromal* stage passing into the stage of *invasion*; (2) a stage of *irritation*; and (3) a stage of *convulsions and coma*. These stages pass more or less gradually into each other and are not always clearly defined. In infants, and occasionally in older children, the onset is often sudden with convulsions. The prodromal stage is sometimes absent and the child is seized with vomiting, convulsions and delirium, and soon dies.

During the prodromal period, between the infection of the meninges and the definite onset of symptoms, the child may show signs of not being quite in its usual health. The mental attributes are altered and he becomes peevish and irritable, dull and listless, and disinclined to talk or play. He mopes and cries without provocation, and is easily tired or unusually drowsy. Mental perversity and hysterical symptoms may be present about puberty. The appetite may be impaired, the tongue coated, and vomiting of the cerebral type may result from apparently very trivial causes. Perhaps there is a little fever. Constipation is often marked; one child, who came under my notice, had been vigorously treated for constipation for a fortnight and even then showed very slight symptoms for meningitis. Diarrhœa is not uncommon in infants. Headache, usually frontal, increased by mental or physical exertion and less frequent in the very young, restless and disturbed sleep, teeth grinding, bad dreams, pavor and enuresis are sometimes present. The duration of this stage varies from a few days to 2 or 3 weeks. Occasionally there is no definite prodromal stage, the initial



convulsions passing rapidly into coma. One child vomited during the day and became unconscious at night. Sometimes the stage is a prolonged one of ill-health, dependent on the predisposing tuberculous disease and incapable of being sharply defined from the onset of the meningeal infection.

Towards the end of this stage the pulse is generally irregular in rhythm and volume, especially on change of position and exertion. It is infrequent, 60-80 per min. in children, 80-100 in infants. Bradycardia is often absent in infants under 1 year of age. Sighing respiration, dryness and loss of elasticity of the skin, and the *tâche cérébrale* are often present. Less frequent symptoms are sucking and champing movements, teeth grinding, abdominal pain with vomiting and fever, and slight delirium at night. In the very young speechlessness is not uncommon, delirium rare.

Gradually the child passes into the stage of *cerebral irritation* with hyperæsthesia of the general and special senses. It dislikes being touched, moved or disturbed in any way; objects to light and noise; does not talk; may complain of the head, crying out "Oh my head," or if unable to talk occasionally gives a sharp piercing cry, the hydrocephalic cry, and is unnaturally drowsy. It lies in bed with the legs tucked up, the abdomen retracted and the trunk flexed; and will sometimes pluck at the bed clothes when they are drawn off. The head is slightly retracted, or the muscles at the back of the neck are more or less rigid, and the head cannot be bent forward without causing pain. Sometimes it cannot be moved from side to side. Rigidity of the neck muscles and retraction of the head are rarely entirely absent. Coarse tremor of the hands and temporary squint may be present. The appetite is bad, constipation persists and is an almost constant symptom, and cerebral vomiting is often present. Sleep is not sound and the eyes are half closed (*ocular catalepsy*). Lethargy deepens, headache gets worse, vomiting more frequent, and sometimes there are convulsions. The pulse and respiration are still irregular, and sighing respiration is not uncommon. Fever is irregular and rarely high. There is incontinence of urine and fæces.

Gradually the child gets worse and worse. The cornea becomes less sensitive and blinking is diminished, leading to lack of polish and slight opalescence of the cornea. Intra-ocular tension is diminished. Variable squint, ptosis, rarely nystagmus, more often conjugate deviation of the eyes and corresponding deviation of the head, may be present. The pupils are often contracted in early stages and later become irregular and unequal, usually dilated and reacting feebly to light and eventually immobile. Choked disc is generally present at the end of the first week, often in one eye before the other, and later on optic neuritis. Carpenter and Stephenson found choroid tubercles in 21 out of 42 cases of acute miliary tuberculosis and tuberculous meningitis; in 8 of the cases in both eyes. They state that these are usually unilateral, small, single, in the form of round, oval or reniform areas of a fawn, grey or paper-white colour, in diameter from



1-3 mm. They are found in the vicinity of the optic disc and macula lutea. Similar tubercles may be found in chronic tuberculosis, even if quiescent. They may be present some weeks before the onset of meningitis, may be quite independent of it, and may become obsolescent. They are evidence of tuberculosis but not of meningitis, and it does not follow that every case of meningitis in a tuberculous subject is due to the tubercle bacillus. Choroid tubercles or optic neuritis were present in 35 out of 52 cases. (Koplik).

The irritability lessens and the child passes through a stage of stupor, during which it can be roused with difficulty and appears rational, into deep coma. Active delirium is rare; coma-vigil is sometimes noted. He no longer speaks or shows a glimmer of intelligence; and frowns little or not at all. The corneal reflex is abolished, and the pupils are widely dilated or immobile (the far-off look). The fontanelle in infants is tense and bulging.

The limbs become rigid, legs extended and the arms flexed or extended. Head retraction is rarely very marked. Spasmodic convulsions set in. They may consist of slight twitchings of the facial or limb muscles, affect one limb only, or be hemiplegic in type or general. They may be more marked on one side than the other, and the distribution may vary during the course of the illness, suggesting that they depend on circulatory disturbance. If they are limited to the same regions throughout, an explanatory patch of intensely distended or thrombosed superficial cortical vessels, of tubercles, or of inflammatory exudation in the corresponding cortical region may be found after death. They may occur at any stage throughout the illness. Temporary palsy of the face or limbs is left after convulsions. Occasionally retraction of the head and opisthotonos become extreme and sometimes there is emprostotonos.

During this stage the child rarely swallows and has to be fed by nasal tube. The lips and teeth are covered with sordes. Vomiting often ceases when the child is fed nasally. Constipation persists and wasting is progressive. The abdomen is often retracted at first, but later on, and even from the onset in infants, it becomes distended from flatulence and intestinal paralysis. The hydrocephalic cry may persist at intervals during the early stage of coma. More commonly it is absent and the child merely utters an occasional low moan.

The pulse rate does not vary directly with the temperature; it becomes more frequent, smaller, and probably more regular. Towards the end it is very frequent, very small and soft, and perhaps uncountable; due to vagus palsy. Vasomotor disturbances appear in the form of brilliant, irregular flushing of the cheeks, sometimes unilateral and perhaps associated with profuse sweating which is also at times limited to one side. The breathing becomes shallow and irregular; noisy and rattling, due to accumulation of mucus in the dorsal posture; and often assumes the Cheyne-Stokes type



during the last few days. This type of breathing usually indicates a fatal issue within 2 days but I have known it occur as long as 14 days before death. It may subside and recur. Periodic grouped breathing with intervening pauses, but without the gradual rise and fall in depth, also occurs. During the pause the pupils are normal or a little contracted; they then slowly and widely dilate, rapidly subsiding with the next pause in breathing. This dilatation is not affected by light. Retention of urine may be due to coma or dulness of the senses, rarely to spinal involvement.

The superficial reflexes are retained until the onset of coma. The knee jerks and plantar reflexes may be normal, absent or exaggerated. At the onset they are often exaggerated. In the stage of coma they are abolished. Kernig's sign occurs in about 60 per cent., less frequently than in other types of meningitis. Babinski's sign is commonly absent, but it may be present after convulsions of the leg and in rigidity. According to Koplik it is more common than Kernig's sign and exceptional in cerebrospinal fever. There is no ankleclonus.

The fever is mild and irregular in type throughout the early stages. The temperature rarely exceeds 100-101° F. and is often about the normal level. It is liable to be modified by active tuberculous disease in the lung or elsewhere, mixed infection or general tuberculosis. It may fall, if previously high from general tuberculosis. From 48-12 hours before death it frequently begins to rise steadily and rapidly, and may go on rising after death even up to 109° F. in the rectum. Sometimes death is preceded by a subnormal temperature. Occasional symptoms are attacks of partial syncope and clonic spasms of the jaw and tongue.

Death takes place quietly from syncope in the stage of coma, sometimes during the act of feeding the child, or during convulsions. Coma may precede death by a few hours or several weeks, and frequently the child lives for 3 or 4 days although death is expected at any moment.

*Varieties in Infancy.*—Three special kinds can be differentiated. In the convulsive type convulsions are followed by fever and rigidity of the neck, with more or less hemiplegia or paraplegia, and then fits ending fatally in 2 or 3 days. A hemiplegic type simulates unilateral brain disease. The paralysis may develop suddenly without loss of consciousness or convulsions. Usually slight fever is followed by hemiplegia and then convulsions; and later on by rigidity of the neck muscles, bulging fontanelle, exaggerated reflexes, coma and death. No explanation of the paralysis is found in the brain or meninges. The somnolent type begins with malaise and vomiting for 7-14 days, and progressive sleepiness which at first is ascribed to fatigue. The child remains drowsy for hours at a time, with the eyes half open. This ocular catalepsy, a fixity of gaze due to absence of winking, depends upon amblyopia and loss of conjunctival reflex. The pulse is irregular, emaciation gradual and progressive, and vomiting,



diarrhoea and tympanites may occur. The drowsiness passes into torpor, coma, and death in 4-8 days after full development.

*Course and Duration* depend very largely on the presence of lung disease or general tuberculosis. Some cases are much more acute than others. Sometimes we find periods of remission or temporary amelioration of the symptoms. They occur in the second stage of the disease and last for a few days up to 2 weeks. The child may regain consciousness and recognise its surroundings, creating false hopes in the minds of the parents and possibly even of the doctor, who at any rate hopes that he has made a mistake in diagnosis. Prolonged remissions for months or years have been noted; 20 cases since 1894 (A. E. Martin, 1909). The symptoms completely disappear. The diagnosis must be confirmed by tubercles in the choroid, or bacilli in the cerebrospinal fluid and the production of inoculation tuberculosis in guinea pigs. Choroidal tubercles are not absolute proof that the meningitis is tuberculous. During the remission the nervous system shows traces of cerebral irritation, indicating that the disease is only dormant. Such cases must be local at first. Ordinarily the progress is slowly but definitely onward to a fatal issue. An analysis of 65 cases showed that 4 died in the first, 27 in the second, 21 in the third, 10 in the fourth, and 3 in the fifth week of the attack. Almost all died in 9-19 days, usually about the fourteenth day. Breast-fed infants live longer than the bottle-fed for nutrition is better maintained. Infants live longer than children because the skull is yielding. Acute cases secondary to tuberculosis of the lungs often end in a few days; convulsions, stupor and coma following each other in rapid succession. Mild cases may last as long as 6-8 weeks. A prolonged period of drowsiness, opisthotonos, rigidity and contracted pupils, may be due to intra-ventricular effusion.

*Diagnosis.*—During the prodromal stage the slight symptoms present may be overlooked or ascribed to some trivial cause such as indigestion, constipation, teething or worms. This is especially true in infants, for dyspepsia and constipation can produce most of the premonitory symptoms. Much stress must be laid upon irregularity of the pulse combined with infrequency, irregular breathing, constipation, vomiting without apparent cause, drowsiness, retraction of the head and convulsions. The main difficulty is in differentiating it during the first two years of life from simple basal meningitis, but in this the symptoms are fewer and milder in type, irregularity and infrequency of the pulse uncommon, constipation often absent, and the fever higher. The age-incidence is of some assistance. In many instances diagnosis is impossible without lumbar puncture. It is also difficult to differentiate it from acute otitis in infancy. An aural examination is necessary. Pneumonia may produce somewhat similar cerebral symptoms but is more suggestive of cerebrospinal or suppurative meningitis. Other causes of retraction of the head, such as described under basal meningitis, must be excluded. The chief error consists in ascribing the onset of the disease to a gastric attack.



*Prognosis.*—It is possible but very improbable that recovery can take place, for the disease is rarely limited to the brain and is usually part of a general tuberculosis. No case can be accepted as one of undoubted recovery unless tubercle bacilli have been found in the cerebrospinal fluid. Reliance upon clinical symptoms for accurate diagnosis is unsafe. Henckel (1900) reported a case of recovery after a long and tedious illness, bacilli having been found in the cerebrospinal fluid. The cases of prolonged remission are also in favour of the possibility of recovery, provided that the disease is limited to a local affection of the meninges. The signs of approaching death are increasing frequency of the pulse, vasomotor paralysis, Cheyne-Stokes breathing, and a rapidly rising temperature.

*Treatment.*—It is of the utmost importance to protect children during the first 5 years of life from exposure to infection. This is particularly necessary after attacks of measles, whooping cough or any lung affection. Once the meninges are attacked we are practically helpless to arrest the disease. The child must be carefully fed, by nasal tube if necessary. Ice or cold by means of Leiter's tubes can be applied to the head, and phenacetin given, to relieve headache. Leeches to the nostrils and mastoids, counter-irritants behind the ears, inunctions of mercury, and many other remedies being tried in vain. Blistering the nape of the neck only inflicts increased suffering; shaving the head merely disfigures the child. Bromides and chloral control the convulsions. Small frequent doses of calomel and iodide of potassium can be given.

The general measures of treatment are devoted to the relief of symptoms and are such as are above recommended in other forms of meningitis (p. 658). The main dependence must be on mercurials and bromides, or chloral, combined with diet, complete rest, and careful nursing.

Operative treatment has been tried. It consists of drainage of the subarachnoid space either by trephining the skull in the occipital region or by lumbar puncture. It is indicated as a means of relieving pressure and on the assumption that the child is dying from coma due to pressure. Such is by no means always the case, for many of these patients die comatose and no increased intracranial pressure is found after death. The coma is chiefly due to the impaired nutrition of the brain, which may or may not be associated with excess of fluid in the ventricles. Moreover, except in very young infants with un-united sutures, we cannot ascertain in any particular case whether there is an excess of fluid in the ventricles or how much, if any, of the coma is due to such excess. Operative treatment must be regarded as experimental rather than curative. The removal of fluid by lumbar puncture can do no harm and may relieve some of the symptoms.

**Syphilitic Meningitis.**—The few instances of supposed syphilitic meningitis are usually of the same type as post-basic meningitis, with much thickening and matting together of the membranes at the base of the brain. At an early age it is impossible of diagnosis from this variety and it is



probable that there is no true syphilitic meningitis in infancy, and that the supposed cases are simply post-basic meningitis in syphilitic infants. A baby, 4 months old, had retraction of the head, moderate fever, wasting and occasional vomiting. There was a family history of syphilis and evidence of the disease in the shape of typical squamous patches on the buttocks and thighs. In spite of specific treatment death occurred in 7 weeks, during which retraction of the head and opisthotonos became extreme, moderate hydrocephalus developed, and vomiting and wasting persisted. A simple basal meningitis was found at the autopsy, with no indication that it was specific.

In congenital syphilis local white thickenings of the meninges and adhesions may be found at the vertex and base, thickening and scarring of the ependyma, and often hydrocephalus. Cortical lesions are a cause of Jacksonian epilepsy. Cerebral syphilis of the acquired type may occur about the age of puberty in the same forms as in adults.

**Pachymeningitis** is rare. It generally depends on contiguous bone disease, e.g. necrosis of cranial bones, disease of the nose or middle ear. It is usually infective. Pus collects between the dura mater and bone, perhaps in sufficient amount to give rise to symptoms of intracranial abscess. Lepto-meningitis and sinus thrombosis are secondary effects. Some cases are syphilitic, even as early as 4 years of age, and associated with gummata or cerebral sclerosis. This variety may be hæmorrhagic, diffuse or basal, or localised, chronic and gummatous. Pachymeningitis of the cord is usually due to tuberculous caries.

*Internal hæmorrhagic pachymeningitis* is occasionally found under 3 generally under 1 year of age. It is due to cachexia, infective diseases, injury, syphilis and hæmorrhagic affections. Sometimes it begins as a serous external meningitis (p. 632) and the hæmorrhage takes place into the effusion secondarily. In cachexia there are no symptoms. Other cases show increased intracranial pressure, bulging fontanelle, headache, vomiting, myosis, papillitis, retinal hæmorrhage, pulse anomalies, twitchings and convulsions. The signs are similar to those of serous apoplexy and the diagnosis is based on the results of lumbar puncture. Some cases are recurrent. It is usually fatal.

*Hæmatoma of the dura mater* is a variety of pachymeningitis hæmorrhagica. It is the result of similar causes, and most frequently due to injury at birth. The common site is the convexity, on one or both sides; and the size is variable. Symptoms vary with the size and locality. They may be absent, localised to one side, or general and like those of the last variety. It may simulate sinus thrombosis. Cases due to injury may recover completely. Occasionally a subdural cyst is left as a sequel. The treatment of these affections is that of the primary disease and directed to the relief of symptoms.



## CHAPTER LI.

### CEREBRAL AND CEREBELLAR DEGENERATIONS.

*Agenesis Corticalis — Cerebral Spastic Paralysis — Infantile Cerebral Degeneration — Amaurotic Family Idiocy — General Paralysis — Cerebellar Ataxia — Auto-intoxications.*

The cerebral and cerebellar scleroses occupy an intermediate position. Some of them are endogenous in origin, due to abiotrophy. Others are exogenous, secondary to encephalitis, congenital syphilis, vascular mischief and other causes. The sclerosis may be limited to the cerebellum or the cerebrum, in whole or in part, or quite localised. The brain substance is shrivelled, brownish and thickened; and shows atrophy and degeneration of the nerve cells and fibres, proportionate to the degree of sclerosis, proliferation of the perivascular tissues and thickening of the vessel walls, and proliferation of the neuroglia, especially in the septa. The initial symptoms are those of the primary disease at first, and subsequently depend on the distribution and extent of the sclerosis. The various affections in which there is sclerosis may be classified according to the date of their origin into :—

#### I. *Pre-natal Group*, due to causes in action before birth.

- (1) Gross cerebral defects, e.g. anencephaly, porencephalus, hydrocephalus, non-development of the prosencephalon and other malformations.
- (2) *Agenesis corticalis*, a backward or arrested development of the nerve cells in the cortex.
- (3) Infantile cerebral degeneration, i.e. degeneration of the cells in the cortex endogenous in origin.
- (4) Delayed development of the motor tract, perhaps dependent on *agenesis corticalis*.
- (5) Diffuse or focal encephalitis or meningo-encephalitis, due to infection or toxæmia of maternal origin.
- (6) Syphilitic endarteritis and thrombosis.



II. *Natal Group*, due to causes in action during birth and almost invariably traumatic.

(1) Meningeal hæmorrhage.

(2) Encephalitis, secondary to injury or primary.

III. *Post-natal Group*; cases arising from causes set in action subsequent to birth.

(1) Arterial disease, endarteritis; embolism, thrombosis and hæmorrhage.

(2) Encephalitis or meningo-encephalitis, due to infection, toxæmia or injury.

(3) Head injury and (?) heat-stroke.

(4) Secondary porencephalus, cysts and tumours.

**Agensis Corticalis.**—A considerable number of cases of diplegic spasticity occur in infants at whose birth there was no difficulty, no injury from forceps, and no asphyxia. Most of these children are small, delicate or premature. Probably there is an imperfect, incomplete, delayed or arrested development of the motor cells in the cortex of the brain or of the motor tract. If this is the explanation, it matters little whether labour was difficult or easy. A possible cause is congenital syphilis but in many cases it can be excluded with certainty. It is difficult to separate agensis corticalis from failure of development of the pyramidal tracts, and probably the two affections are combined. If the cause is in the cortex it is likely to be generalised and not limited to the motor regions. The mental capacity will be considerably affected. A mere delay in development of the pyramidal tracts will not affect the mental state.

The pyramidal tract is developed in the fifth to the seventh month of foetal life, and in premature infants is almost wholly non-medullated and therefore functionless, for the myelin sheaths of this tract develop at the end of the ninth month and the tract is not fully developed until later. There is the further possibility that, because of the incompleteness of its development and lack of protective myelin sheaths, it is more liable to injury during labour. In its downward growth during infancy the lowest fibres of this tract receive their myelin sheaths last and may never acquire them. For this reason the spastic paralysis is most common and most permanent in the lower limbs.

**Cerebral Spastic Paralysis.**—*Syn. : Little's Disease—Congenital Spastic Rigidity of Limbs—Congenital Birth Palsy—Spastic Paraplegia—Infantile Spasmodic Paraplegia—Primary Spastic Paraplegia—Cerebral Diplegia—Paraplegia Spastica Cerebralis—Spasmodic Spinal Paralysis—Spasmodic Tabes Dorsalis.*—Heine described this disease in 1840. Little fully investigated it during the years 1846-1870, and in 1862 published a paper



“ On the influence of abnormal parturition, difficult labour, premature birth and asphyxia neonatorum on the mental and physical condition of the child, especially in relation to deformities.” He first suggested the relation of the symptoms to injury of the brain at birth. The affection Little described was spastic paraplegia due to meningeal hæmorrhage. Since then the name “ *Little’s Disease* ” has been confined to cases occurring in infants born before term or at term with instrumental assistance. In a wider sense “ cerebral spastic paralysis ” includes cases in which there is evidence of an affection of both cerebral hemispheres, and those of spastic palsy and rigidity due to a unilateral cerebral lesion. The characteristics of these diseases are similar but their localisation varies. Post-natal cases are more likely to be unilateral than ante-natal and natal ones. Rigidity is the chief feature, with or without associated symptoms. According to their distribution and characters they are divisible into :—

- (1) Hemiplegia. Monoplegia with contracture. Spasmodic facial palsy.
- (2) Cerebral diplegia or bilateral spastic (spasmodic) hemiplegia.
- (3) Affections in which rigidity is slight ; viz. bilateral and unilateral athetosis, athetotic diplegia, congenital spastic chorea.
- (4) General spastic (spasmodic) rigidity with varying degrees of idiocy. Atypical forms occur in which the mental changes are associated with slight general rigidity, the mischief being mainly in the frontal region.
- (5) Paraplegic rigidity or Little’s disease. Paraplegic paralysis is a rare sub-variety.
- (6) Familial spasmodic affections.

In all these affections we find persistent rigidity, an intact reflex arc, and alterations in the pyramidal tract which are generally cerebral, occasionally spinal, in origin.

The motor residua of cerebral palsies are of several kinds. In some instances they are so marked that the disease is named in accordance with the preponderance of a special residuum, as is seen in the following table of the different varieties :—

- (1) Contractures.
  - (a) Spasms :—clonic, tonic, or intermittent.
  - (b) Muscular rigidity.
  - (c) Early contractures :—paralytic, passive and temporary.
  - (d) Late contractures :—constant, continuous and fixed.



- (2) Exaggerated tendon reflexes.
- (3) Associated movements.
- (4) Tremors :—(a) Simple, like those of paralysis agitans or of disseminated sclerosis ; (b) Reflex clonus.
- (5) Hemichorea, simple and constant.
- (6) Hemi-ataxia, a disturbance of co-ordination on intended movements.
- (7) Athetosis.

Out of 859 collected cases (R. Jones, 1906) the percentages of the different kinds of paralysis were hemiplegia 60, diplegia 18, paraplegia 16, monoplegia 4.

*Pathology.*—The causation of these affections is illustrated in the classification of the various groups. Maternal illness, eclampsia, and injury during pregnancy may have some predisposing influence. Of natal cases the majority are due to meningeal hæmorrhage from injury by too narrow a pelvic outlet, forceps, or asphyxia due to constriction of the neck. The child may be born black in the face, have convulsions during the first weeks of life, and be mentally deficient. When forceps are used the damage is probably due to the defective mechanism of labour, e.g. a large head and small pelvis, which necessitates their use, rather than to actual injury by the instrument. Some cases are due to violent precipitate labour. In view of the position of the motor areas it is easy to understand that hæmorrhage on the surface of the brain may occur in such a position as to affect the leg centres only. According to the extent and the direction in which extension takes place, it is obvious why in some instances one or both arms, and even the face, are also involved. If it extends forward on to the frontal region, the mental faculties will be impaired. The larger the hæmorrhage the wider is the distribution of the spastic paralysis and the greater the probability of mental impairment. If it spreads to the occipital region it may cause squint and hemianopia. The hæmorrhage is usually under the arachnoid on the convexity of the cerebrum, generally bilateral, and diminishes in extent from the median line outward. Less frequently it is between the arachnoid and the dura mater, and occasionally subdural. It is due to compression of the superior longitudinal sinus and its veins from dislocation of the cranial bones during labour, or to raised pressure from asphyxia. These infants are often born dead or die soon after birth. Atelectasis is generally present. Subarachnoid hæmorrhage causes most damage to the nerve tissues. Encephalitis or meningo-encephalitis may follow on the injury, or be due to infection or toxæmia before or after birth. Both these affections produce secondary sclerosis. An acute onset, with fever, and a subsequent tendency to improvement, are in favour of germ infection. Congenital hemiplegia is due to unilateral cerebral defect, or to endarteritis



and thrombosis of the middle cerebral artery, or syphilitic disease of the veins entering the superior longitudinal sinus.

Post-natal cases are generally hemiplegic and the result of focal encephalitis, embolism, thrombosis or hæmorrhage. Quite three-fourths begin under 3 years of age. They occur during the course of infective diseases (p. 697), and are often ushered in with one or more fits, which may prove fatal. Possibly convulsions may cause the local mischief, if a hæmorrhage, but they are more likely to be the result thereof. As the child recovers the paralysis becomes obvious and the subsequent occurrence of sclerosis engenders the spasticity. When a general sclerosis results, as after a general encephalitis, the sclerosis and its effects resemble those of natal and ante-natal causation and can only be distinguished by the history, that is, they date in onset from some acute illness after birth.

*Morbid Anatomy.*—Atrophy of the cortical regions of the brain is always present, and more or less sclerosis, with or without porencephalus. In pre-natal cases the pyramidal tracts are undeveloped; in post-natal ones they show degenerative changes. In recent natal cases extravasations of blood over the cortex, and lacerations of the cortex of the cerebrum and cerebellum, have been found. Gross lesions are characteristic of the hemiplegias and general sclerosis of the diplegias. Circumscribed atrophic sclerosis, superficial shrunken patches, or cysts may be found. Osler states that cerebral sclerosis is found in 50, porencephalus in 24, meningeal hæmorrhage in 9, and embolism in 7 per cent. The cerebellar cortex may be much atrophied. In congenital syphilis the pia-arachnoid is thickened and the cortex atrophied.

The cell degeneration may be primary, due to the toxæmia of syphilis or some infective disease. Or it may be secondary to pressure from hæmorrhage, thrombosis of the veins entering the superior longitudinal sinus, embolism or thrombosis of the Sylvian artery, or multiple intra-cerebral hæmorrhage from asphyxia neonatorum. Microscopical examination may reveal degeneration of the large pyramidal cells; old or recent degeneration, or absence, of the pyramidal fibres; and degeneration of the direct and crossed pyramidal tracts.

*Symptoms.*—Males are more liable than females because they are larger at birth. In ante-natal and natal cases the affection usually shows itself during the first few days of life as a stiffness of the limbs which gradually becomes more pronounced. In very mild forms, and among the unintelligent, it may not attract attention for several months or not until the normal child is expected to try and walk. The adduction of the lower limbs may be the first sign to attract notice and may not be observed for several months. The child is a reflex animal at first and myelinisation of the cord is not complete until the end of the first year. Post-natal cases are generally of sudden onset in the first 3 years of life, during the course of an acute illness, ushered in by fits, and hemiplegic. Pre-frontal sclerosis may



give rise to impaired intelligence, asymmetry of the cranium, slight general rigidity, and backward speech if it is on the left side. The symptoms vary with the site of the lesion. Palsy and spasm are generally unequally combined.

In a marked case of diplegia there is great rigidity, chiefly of the extensor type, and paralysis. The rigidity preponderates. The head and upper part of the trunk are inclined forward and move as if inseparable. Backward extension and lateral deviation of the head are due to involvement of the neck muscles. The arms are applied to the trunk; the forearms flexed; the hands pronated, flexed at the wrists and inclined toward the ulnar border, and the fingers and thumbs may be tightly clenched. Sometimes there is extension or hyper-extension of the forearms and hands. Movements are awkward, slow, and accompanied by rigidity. Grasping, supinating and throwing are performed with difficulty. In marked contractures movements are impossible and even passive ones are violently resisted. In contra-distinction to spinal palsy the loss of power is greatest in the hand. The lower limbs are rigid, rotated inward, and slightly flexed at the hip and knee. The thighs are strongly adducted as far as the knees and sometimes crossed. Below the knees an oval space is formed, owing to the inward rotation of the limbs. The feet tend to equinus, because of contraction of the gastrocnemii. The back is hollowed.

The rigidity may be so great as to render sitting impossible. In milder cases, on sitting, the legs are held rigidly extended on account of the flexor rigidity at the hip joints. On standing it is usual for the plantar surface of one foot to rest on the instep of the other. Movements are slow and clumsy. The typical gait is cross-legged and spasmodic. Often the child is unable to walk at all or, even when supported, can only make very imperfect efforts on tip-toe, with short rigid steps and the knees closely pressed together. The point of the foot is drawn along the ground with a movement of circumduction and comes to rest on the other foot, or is carried forward and across in a "cross-legged" manner. All these movements may be precipitate because of a kind of foot clonus. Other muscles are more or less involved. Those least under the influence of the will are the most likely to escape. Spasmodic rigidity and an expression of stupidity depend on implication of the facial muscles. *Risus sardonicus* has been noted in one instance. The legs are most and the face least affected.

The eyes are usually unaffected unless the face muscles are implicated. Squint, generally convergent, is due to spasm or hypermetropia. Inequality of the pupils is not uncommon. Nystagmus is unusual. Congenital cataract, primary bilateral optic atrophy, and palsy of the third and sixth nerves have been noted. Vision is usually good. Bulbar symptoms may be present. The tongue may be protruded slowly, with difficulty or not at all, or may deviate to the sound side. Difficulty in swallowing is due to spasm of the pharyngeal and oesophageal muscles. The child may only be able to take



liquid or semi-solid food for some years. Slobbering and difficulty in chewing may be present. Speech is often affected. It is slow and drawling, or jerky as if the words "emerged with difficulty." Sometimes there is aphonia, stuttering or dumbness. The child is backward in learning to speak and express ideas in words. The defect is mental or due to rigidity and articulatory difficulty. Crying is hoarse, and prolonged if there is general rigidity.

The rigidity diminishes during sleep and is increased by fear and other emotions. There is no true muscular atrophy. The affected limbs or limb may be abnormally small from non-development, due to disuse and deficient innervation. Sometimes the muscles seem actually hypertrophied and are unduly hard. There is no sensory disturbance. Intention tremor is present in mild cases.

All the deep reflexes are greatly exaggerated, though there may be difficulty in obtaining them because of the rigidity. The periosteal reflexes are marked. Ankleclonus, patella and jaw clonus may be present. The plantar reflex is extensor in type. Superficial reflexes are lessened, normal or increased.

Electrical irritability is normal or increased. In rare cases tetanoid contraction has resulted from faradism. There is no alteration in muscular sense, vasomotor functions, or the functions of the bladder and rectum, except a tendency to spasm in some instances. At times the affection is so slight that it attracts no attention.

Post-natal hemiplegia is essentially the same as in adults except in its onset. The damage may be slight, transient, or last for a few months and end in complete recovery. It is rarely complete ultimately. The grasp may be as strong as on the sound side but the ordinary performance of voluntary movements is interfered with. There is a variable degree of rigidity associated with a variable amount of mobility in the form of mobile spasm, athetosis, chorea or tremor, usually the intention tremor of disseminated sclerosis and known as chorea spastica. The leg may recover completely or exhibit mere clumsiness, a liability to trip and fall, spasticity, or a varying degree of athetosis. The nutrition and development of the affected limb are much interfered with. Hemiplegia is rather more common on the right side. Initial monoplegia is rare and apparent monoplegia is generally due to partial recovery. Palsy is at its maximum in the second week.

There is not much palsy of the face and trunk. The arm is more affected than the leg, and the distal segments more than the proximal ones. There may be a little weakness on the same side as the lesion because of some "uncrossed fibres." The facial palsy is most apparent on laughing and crying and may be associated with exaggerated or choreiform movements. It may not clear up though the arm and leg recover. The limb is often reddish-blue in colour, possibly from vasomotor paresis. Contractures and



distortions result in bad cases. Chorea and athetosis may cause muscular hypertrophy. Aphasia is most frequent in right hemiplegia. If due to the depression of acute illness or general cerebral disturbance it is evanescent. It is indicated by backwardness in learning to speak. Except in bilateral lesions or one on the left side in later childhood it is temporary, for the speech centre on the opposite side undertakes the functions of the one destroyed or damaged.

In these post-natal cases the damage may be too slight to cause permanent palsy, or be situated outside the motor area. In either case there is a tendency to fits. Slight degrees of paralysis are liable to be overlooked in infancy. The nerve elements in the damaged cortex are softened and destroyed. Secondary scar tissue forms and fits are set up by irritation, produced by the slighter damage in the immediate neighbourhood of this local area of impaired vitality. They are Jacksonian in type but may become generalised, by overflow to the surrounding cerebral tissues.

In all cases of cerebral sclerosis the mental state must be carefully noted for on this depends the prognosis. Except possibly in agenesis of the motor tracts and cases in which the damage is limited to the motor area there is always some mental impairment. The child may be intellectually normal, merely a little feeble-minded or deficient, or partially or completely idiotic. There are many gradations. Placidity is common but the child is often silly, odd, capricious, shy, timid, impulsive, disagreeable, irritable, destructive, vicious, violent and dirty. Even in the mildest cases there is as a rule some defect but it may not be noticed until he enters on school life and into competition with other children.

*Complications* are limited to fits and various deformities, such as asymmetry of the head, facial hemiatrophy, microcephalus and hydrocephalus; retraction or over-flexion of the head and torticollis; kyphosis, lordosis and scoliosis; unequal scapulæ and pelvic bones; equino-varus and pes cavus; and various forms of tremor, chorea and athetosis.

*Diagnosis.*—Cerebral sclerosis must be distinguished from tetanus neonatorum, infantile palsy, amyotonia, myotonia, transverse and compression myelitis, Friedreich's disease, disseminated sclerosis, endogenous myopathies, cerebral syphilis and hysteria. A double lesion in the brain might cause double infantile spastic hemiplegia but must be extremely rare. Most of the above diseases can be differentiated without much difficulty. Chief stress is laid on the spasticity, exaggerated reflexes, mental state, and the past history. Post-natal cases are generally hemiplegic and of sudden onset. A mild diplegia may become hemiplegia through the effects clearing up on one side. Slight degrees of rigidity and mere clumsiness are not always recognised. Athetosis indicates incomplete destruction of the motor centre or an attempt at spontaneous cure.

*Prognosis.*—Few severe infantile cases survive 5 years. If they do, they may live beyond puberty. In cases of mild or medium severity the



outlook as regards life is good, but as regards recovery it depends primarily on the cause and secondarily on the duration and extent of the rigidity and the mental state. If it is due to delayed development of the motor tract, the child will gradually improve and though for years he may be backward in learning to talk, walk, play games and do mental work, he may eventually differ in no respect from the normal boy. Complete acquirement of motor power and skill in the use of the muscles is rare. The child commonly betrays some clumsiness. His mental backwardness depends on the delay in teaching or in learning to speak properly. Some of these children stutter badly. A few are mentally precocious. The rigidity may disappear entirely; first from the face and neck, then from the upper extremities, and finally from the lower ones, in which however it may be permanent. Mental imbecility may be present though the other signs clear up. If so, there is probably cortical agenesis as well as delayed development of the motor tracts. Puberty is not delayed. The disease is not transmitted to children.

When secondary to meningeal hæmorrhage, the prognosis depends on the amount of the hæmorrhage and its effect on the mental state. In fact, whenever the cause is not one of mere delay in development, the outlook depends chiefly on the degree of mental incapacity, and is progressively worse as this approaches idiocy. Many improve a little and then remain stationary. The prenatal cases rarely exceed 30 years of age, and the post-natal ones are often progressive and fatal within 3 years.

In the post-natal type recovery is rare, if rigidity has appeared. Even if the palsy clears up, there is some impaired development of the affected side. Mental acuity is often subnormal. In unfavourable cases athetosis, Jacksonian epilepsy, epilepsy and, less often, intentional tremor may arise. The paralysed limb does not grow properly, remains small, and may lead to limping and scoliosis. In cerebral monoplegia only one limb is permanently affected. Epileptic fits sometimes appear at the onset and remain prominent to the end, but they may cease, or may develop later. They are liable to increase in frequency, and may end in status epilepticus or sudden death. Usually death is due to malnutrition, epilepsy or intercurrent disease. Epilepsy does not reduce the possibility of mental and physical improvement, but increasing age does very considerably.

*Treatment.*—Rely chiefly on warm baths followed by methodical education of the limbs by passive movements, massage, voluntary movements and gymnastics. Massage, galvanism, passive movements and warm garments assist in maintaining nutrition until voluntary power develops. It may be advisable to tie up the sound limbs to encourage the use of the affected ones. Heavy boots are injurious. Dividing the tendons of spastic muscles relaxes the spasm of adjacent ones as well. A flexed wrist must be hyperextended, with the thumb at right angles to the palms, for 1-2 years. Tenotomy and tendon transplantation are of value. Remedy deformity



which limits the usefulness of the limb. Operative treatment is useless if paralysis is complete and spasm constant, and in cases of idiocy, microcephalus, athetosis and (?) epilepsy. Careful education of the speech, special senses and mental functions is essential. These children must be encouraged, not bullied. The mode of life should be that of ordinary children as far as possible. Success depends on and justifies prolonged systematic treatment.

**Infantile Cerebral Degeneration.**—*Syn.: Diffused Cerebral Sclerosis.*—This is probably also identical with the “family chorea and hereditary family athetosis” of Massalongo and Oppenheim, and with a sub-variety “family ophthalmoplegia.” It is an endogenous tendency to premature failure of nutrition and function of nervous structures, and must be distinguished on etiological grounds from sclerosis due to encephalitis or congenital syphilis. Possibly some cases are terminations of interstitial encephalitis. Anatomically there is found extreme cerebral atrophy, sclerosis of many convolutions and especially of the white matter and basal ganglia, some cerebellar atrophy, degeneration of the pyramidal tracts and the ascending cerebellar tracts, and isolated patches of sclerosis. The nerve cells are abnormally small.

It generally begins in the first or second year of life. An apparently healthy child, if able to walk, begins to show signs of difficulty in walking. This increases and is associated with spasticity, exaggerated reflexes, and a similar defect in the upper limbs. It may start in the legs or in all four limbs. The muscles of the face and mastication become involved. The movements are sluggish and awkward. There is inability to carry out purposive movements, increased muscular tone, rigidity and contractures. Tremors, ataxia and athetosis are not uncommon. The muscles are tense and board-like. There is no true palsy or atrophy, and no change in electrical reactions. Squint, nystagmus, conjugate deviation, choked disc and optic atrophy, and pseudo-bulbar symptoms have been noted. The pupils react. The power of speech is rapidly lost. The mental state is rarely unimpaired; usually there is increasing idiocy. The disease is progressive, spasticity becomes extreme, distressing convulsions occur, and death results from emaciation, bedsores or intercurrent disease. The above symptoms are combined in various ways in different cases according to the localisation and extent of the sclerosis.

Higier (1897) reported *progressive ophthalmoplegia externa* in 4 sisters. The symptoms were like those of cerebral sclerosis but the onset was late, beginning with weakness and spasticity in the legs at 7-12 years of age. It gradually progressed to spastic paraplegia and talipes equino-varus, inability to stand, walk or sit, tremor and clumsiness in the hands, defective speech, impaired intelligence, squint and optic atrophy. It affects both sexes and is transmitted by either. Sporadic cases are rare. In other instances the levator palpebræ superioris or the abducens, perhaps in conjunction with the facial nerve, was affected.



*Infantile progressive bulbar palsy* begins at 6-10 years of age. Speech, swallowing and expression are affected. Dribbling of saliva is followed by atrophy and tremor of the tongue, a mask-like aspect, palatal palsy, weakness of the muscles of mastication, irregularity of the pulse and breathing. It may remain stationary for some time.

**Amaurotic Family Idiocy.**—*Syn. : Primary Amaurotic Mental Deficiency (Warren Tay)—Infantile Degeneration with Changes at the Macula—Arrested Cerebral Development—Family Form of Idiocy—Agenesis Corticalis.*—This is a variety of cerebral sclerosis or degeneration described first by Warren Tay in 1881. In 1901 Falkenheim collected 64 cases, of which 27 were isolated ones and 37 occurred in 13 families. Four of the 36 families were Gentiles. A good many cases have been reported since.

The disease is almost peculiar to Jews, especially poor Polish and Russian Jews. It occurs in families, affecting several children in no regular sequence. No etiological factor has been found. The children are usually normal at birth and breast-fed. It begins between the second and tenth months, generally at 6 months of age, with weakness of the muscles of the neck and back, inability to support the head, dull expression and imperfect vision. This is followed by apathy, imbecile aspect, increasing weakness, acuteness of hearing, and perhaps persistent screaming. Perception of light is left and the electrical reactions are unchanged. The paralysis is flaccid or spastic, or spasticity may follow flaccidity; the reflexes are normal or increased. In late stages there is atrophy of the enfeebled muscles, retraction of the head, rigidity of limbs, progressive emaciation and idiocy. Death results from marasmus or intercurrent disease. At birth the eyes are normal. Haziness in the macular region is the first sign; then the macula becomes occupied by an oval whitish-grey, bluish-white, greenish-white or dead white patch, about twice the diameter of the optic disc, in the centre of which is a round cherry-red spot, the fovea centralis. These changes remain stationary. The optic disc may show neuritis or atrophy, and the surrounding pigment disc is very distinct. Unequal pupils, squint, ptosis and nystagmus are sometimes present. The pupils usually respond to light.

Hyperacusis is not always present. Some children appear deaf. Spasms or fits may be induced by sudden noises. Explosive laughter and dysphagia or disturbance of deglutition have been reported. The terminal symptoms suggest meningitis, and death may be unexpectedly sudden. Out of 38 cases (Falkenheim) 35 died under 2 years and the other 3 under 4 years of age. The characteristic ocular changes are not found in all cases.

The essential change is a degeneration of the ganglion cells of the retina, and of the optic nerves and tracts. In McKee's case Spiller found the ganglion cells of the retina increased in size, more globular, and much degenerated. At the fovea centralis there are no such cells and the red choroid is visible; but at the margins of the fovea the ganglion layer is



6-10 cells deep and, being swollen, gives rise to a white area round the central red spot. These cells are genetically part of the central nervous system. Degenerative changes were found in the optic nerve and the nerve cells throughout the spinal cord. Hirsch first found these extraordinary cellular changes in the retina. Spiller could not find a single normal cell throughout the central nervous system. The disease is not inflammatory, and must be regarded as a primary degeneration of nerve cells and secondary degeneration of nerve fibres; varying in distribution. It differs from infantile cerebral sclerosis in the cellular changes.

Possibly these cellular changes are compatible with more prolonged life. More probably cases in older children are different. They exhibit *progressive mental degeneration and changes in the fundus*. A normal child, at 3-14 years of age, becomes irritable, bad tempered and mentally dull, perhaps timid and emotional. The ocular symptoms are defective sight, loss of central vision, moderately dilated pupils which react badly to light, perhaps nystagmus, pallor of the discs, and mild choroido-retinitis in the macular region. Spasticity, inco-ordination, and absent or deficient knee jerks, may be present. The loss of central vision induces a crab-like gait. It progresses to blindness and imbecility. Some improve on pot. iod.

A family form, analogous to *family spastic paraplegia*, begins about the eighth year of age. It is very slowly progressive, with gradual development of spasticity, and is possibly due to overwork of the neurons at the time of functional activity.

**Juvenile General Paralysis.**—This is due to congenital syphilis, though the signs thereof may be absent in the patient yet present in other children. It affects both sexes equally. It begins about puberty, sometimes as early as the eighth year. A definite family history of syphilis is found in 60-80 per cent. of the cases, and Mott states that general paralysis occurs in the father in 20 per cent.

Mott describes two types. In one there is an arrest of development, and the child is infantile in body and mind. In the other there is fair physical and mental development until puberty or adolescence, and then progressive decay of the central nervous system. The degeneration of body and mind may be started by the strain of school life, puberty, accident, acute illness or spontaneously. In a third type the child is unduly sharp and intelligent.

The early signs are an aspect of worry or pre-occupation, apathy, loss of memory; outbreaks of crying, passion or screaming, symptoms of cerebral irritation; attacks of partial paralysis, of mental stupor or unconsciousness, or epileptiform convulsions. The pupils are unequal, react sluggishly or not at all to light, and optic atrophy may be present early. Fine tremors of the tongue, facial muscles and hands are present. The knee jerks are exaggerated early and lost eventually. Speech is indistinct, slovenly, slow or rapid; and sometimes ceases. Delusions are



uncommon and megalomania rare. Childish ideas of grandeur, such as might be expected at the particular age, occur in about 10 per cent. Sometimes there are attacks of excitement and depression.

The course is steadily progressive physical, mental and moral failure without remission. Weakness of the limbs is followed by paralysis and contractures. The face is expressionless. Sucking, chewing, humming and slobbering occur. The sex organs undergo fibrosis and there are no spermatozoa, Graafian follicles or catamenia. The testes may be undescended.

*Diagnosis* is often difficult. Many cases are mistaken for idiocy or a congenital idiot may develop the disease. The exaggerated reflexes, sluggish light reflex, and early occurrence of paralytic attacks are the chief indications. Evidence of congenital syphilis may be present, e.g. scarring at the angles of the mouth, interstitial keratitis, choroidal atrophy or deafness. The chief difficulty is the differential diagnosis from endarteritis of the brain of specific origin. It is important with a view to prognosis. Fits may simulate epilepsy.

The average *duration* is 2-4 years, varying from 6 weeks to 7 years. It is invariably fatal, and a chronic diffuse meningo-encephalitis has frequently been found after death. It is not a post-syphilitic degeneration entirely.

**Cerebellar Ataxia.**—The cerebellar cortex has an important influence on the movements of skeletal muscles. Luciani found that ablation of the cerebellum caused general unsteadiness, strabismus, spasms of the neck muscles, flexions of the fore-limbs, hypotonia, and unsteadiness of the posterior limbs; the symptoms passing off in a few months. If the cortex was damaged in the region of the sigmoid gyrus, the symptoms persisted. Therefore, atrophy and even complete absence of the cerebellum can exist without symptoms. Mild cases of disease may recover. Injury to the cortex is indicated by muscular rigidity, reflex and mental symptoms.

Marie and Sanger Brown have described *Hereditary Cerebellar Ataxia*. It occurs in several members of a family in association with a congenital cerebellar defect. It begins in the legs and then affects the arms, face and speech. The gait is reeling, rather than ataxic; the Argyll-Robertson pupil is sometimes, nystagmus rarely, and optic atrophy often present; the knee jerks are exaggerated always; and there are articulatory difficulties. It is closely allied to Friedreich's disease (p. 715), and comes on at 15-35 years of age. Cerebellar hypoplasia and secondary degenerations are found after death.

*Cerebellar Ataxia or Diplegia* is sometimes an ataxia of congenital origin starting in early life. Batten recognises three types:—(1) Noticed from earliest life and tending to improve. (2) Of sudden onset during acute illness, and perhaps ending in recovery. (3) Of gradual development in a normal child, progressive, and of bad prognosis. The symptoms are



unsteadiness of the head, trunk and limbs ; unsteadiness in sitting, standing and walking ; slow monotonous, drawling, sing-song, indistinct articulation ; slowness or difficulty in swallowing. Intelligence is unimpaired ; the habits are clean ; there are no eye symptoms, and no rigidity. Cases must be diagnosed from ataxia due to tumours of the cerebellum or mid-brain and sometimes seen in diphtheritic palsy ; from cerebral diplegia with associated choreiform movements or ataxia ; from Friedreich's disease and disseminated sclerosis. Some of them are mixed cases of cerebral and cerebellar mischief. In these there is usually rigidity, mental impairment, exaggerated knee jerks and extensor plantar reflexes.

The disease is rarely progressive and tends markedly to recover. A child ataxic and unable to walk at 2 years of age may walk steadily in another 2 years. Treatment is directed to improving co-ordination by the use of a solitaire board, mozaic puzzles, etc., for the hands ; Fraenkel's exercise board, walking and various combined movements for the legs.

**Functional Familial Disorders.**—Though not degenerative in causation these rare affections present analogies with the above diseases which render it convenient to consider them in connection therewith.

*Family Periodic Paralysis* was first reported by Hartwig (1874) as "intermittent spinal paralysis." Up to 1907 about 85 cases were on record. It is characterised by periodical attacks of motor paresis or paralysis, flaccid in type, with weakness or abolition of the reflexes, and partial or complete loss of electrical excitability. Sensation is altered. It may affect several members in the same family. It begins at 6-24 years of age. F. Buzzard has reported a case starting in infancy.

The attacks come on gradually during a period of rest after physical over-exertion, usually in half an hour or so and often during the night. They may be preceded by a sense of fatigue, "pins and needles" over the trunk and limbs, or a dull aching sensation in the legs and a sinking sensation in the epigastrium. The severity varies from slight temporary paresis up to complete immobility ; the duration from an hour to days ; and the frequency from daily ones to attacks at prolonged intervals. All the voluntary muscles are affected, especially the proximal limb muscles. It appears in the legs, arms, trunk and neck successively. Except in severe attacks the muscles of respiration and those supplied by the cranial nerves escape. The diaphragm is never affected and breathing is entirely diaphragmatic. There is no sphincter trouble, but weakness of the abdominal muscles interferes with micturition and defæcation. Speech and swallowing are affected.

The superficial and deep reflexes are abolished or diminished. There is a gradual reduction in the response to mechanical, electrical and reflex stimulation, in proportion to the loss of voluntary power. The heart often dilates, is irregular, and may present a systolic murmur. Sensation and consciousness are unimpaired. Sweating is often present.



Buzzard ascribes it to stagnation of lymph in the muscular lymph spaces, and some consequent effect on the muscle plasma. In support of this view is the fact that many patients can walk off the attack; and the escape of the diaphragm and the muscles supplied by the cranial nerves, for they are never so completely at rest as the limb muscles and therefore do not suffer from lymph stasis. Indiscretions in diet and constipation are predisposing causes, and increased toxicity of the urine has been noted, suggesting auto-intoxication. Possibly the two factors are combined in action.

Recovery takes place in inverse order to the onset. The disease does not shorten life. Attacks become less severe, less frequent, and may cease as age increases. Syncope may occur from the cardiac dilatation. Treatment should be directed to the prevention of toxæmia and auto-intoxication.

*Myotonia Congenita or Thomson's Disease*, is a spastic contraction of the muscles coming on after sitting. After much effort the patient is able to walk and as he walks the movements improve. It affected 12 out of 14 in Thomson's family. Mikinoff (1897) collected 100 cases. It affects more males than females and is transmitted by either sex. It has been observed in infants at the breast in the form of inability to open the mouth, immobility of the face on beginning to cry, inability to nurse, frequent sighing respiration, interference with movements of the limbs, and hypertrophy of the leg muscles.

The chief signs are impediment in gait, temporary rigidity at the onset of effort, and the disappearance of rigidity with return of suppleness of the muscles. They are increased by fatigue, damp, heat, cold and observation. All the muscles may be involved, even the eyes and tongue. The spasmodic state is universal but most in the legs, arms and face. Muscular development is very marked; knee jerks active; and a localised muscular swelling is induced by a sharp tap. A very small faradic current induces tetanic contraction, or clonic contractions which persist after the current is turned off. The muscles are easily excited by galvanism, and the reaction of degeneration has been noted. This disease has been ascribed to auto-intoxication. If so, it is due to a poison which has exactly opposite effects to the one which causes family periodic paralysis. It runs a prolonged course, with periods of remission and improvement.

**Landry's Paralysis.**—Few cases occur in children. It is an acute intoxication affecting the nerves, cord or bulb, and causing polyneuritis, neuritis of spinal nerve roots, poliomyelitis or bulbitis. It may begin with bulbar symptoms. Generally it starts in the feet and affects the bulbar region finally, causing death from asphyxia without loss of consciousness. The superficial and deep reflexes are absent and electrical irritability disappears. The sphincters escape and there are no trophic effects. It is almost always fatal in 5-10 days.



**Treatment.**—In all the organic affections described in this chapter treatment is of comparatively little value for curative purposes. It must be directed to maintaining the general health, conserving strength, and relieving symptoms. Anti-syphilitic remedies must be tried if there is the least possibility of a syphilitic causation.



## CHAPTER LII.

### VASCULAR AFFECTIONS—TUMOURS—ABSCESS.

*Concussion—Intracranial Hæmorrhage—Thrombosis—Embolism—Cerebral and Cerebellar Tumours and Abscess.*

**Concussion** of the brain, if severe, causes “bruising” and hæmorrhagic extravasation. It is infrequent in children and rare in babies, because they do not often fall from any great height and the yielding bones allow displacement of the brain. Concussion gives rise to loss of consciousness, vomiting, infrequent pulse and retention of urine. In actual bruising of the brain there may be aphasia, transitory palsy, and signs of cerebral irritation, shown by irritability, refusal of food, vomiting, and the curled up attitude in bed with resentment at being disturbed. Mild cases of concussion recover in a few hours or days. Severe ones may be fatal. It is advisable to treat even the mildest case with rest, quiet and careful feeding, for it is impossible to make an accurate estimate of the amount of damage and time must be allowed for its repair.

**Intracranial Hæmorrhage.**—The bleeding is meningeal or intra-cerebral. Meningeal hæmorrhage is usually vertical, involving one or both convexities of the cerebrum, and may extend to the base and cerebellum. It is commonly due to injury at birth by narrow maternal passages, forceps or prolonged constriction of the neck. The overlapping bones compress the veins of the longitudinal sinus. It may occur in normal labour, especially if premature. After birth it results from injury, pachymeningitis (p. 670), pertussis, the hæmorrhagic types of specific fevers, sinus thrombosis, and various blood-states such as scurvy, purpura, leukæmias and hæmophilia. In some of these the bleeding takes place under the dura mater, causing a subdural hæmatoma (p. 670); generally it is from vessels in the pia-arachnoid.

Intra-cerebral hæmorrhage may be intra-ventricular from the choroid plexus or into the substance of the brain. Occasionally it occurs in apparently healthy children and no cause can be found after death, e.g. into the ventricles at 2 months (G. Carpenter), left temporo-sphenoidal lobe at 19 months (Murray), pons at 6 years (Collier), cerebellum at 9 years (H. H. Phillips), frontal lobe at 10 years (Edwards). The usual causes are the same as in meningeal hæmorrhage, or malignant endocarditis and embolism, occasionally atheroma, arterio-sclerosis, aneurysm and syphilis.



*Symptoms.*—At birth the signs are often those of asphyxia only (p. 116), subnormal temperature and cyanosis. Sometimes there are convulsions, unequal pupils, shallow irregular breathing, and slow pulse. Or there may be no asphyxia, but fits and inequality of the pupils come on in a few days. Fits are more or less unilateral when late in onset. Trismus, spasms, exaggerated reflexes, ocular palsies, irregular pulse and breathing, and increasing coma or palsy are sometimes present, and probably due to increasing effusion. Usually there is no evidence of paralysis. The fontanelle bulges and does not pulsate. Generally speaking the onset is with convulsions or coma, and the sequel is spastic paralysis. In older children the symptoms are the same as in adults, viz. headache, coma, delirium, convulsions, palsy, etc. The attack may begin with convulsions.

*Diagnosis* is important in cases due to injury at birth, because of the possibility of relief by operation. It is based on the history of prolonged labour, use of forceps or asphyxia, and the fits or coma with bulging fontanelle. Trephining is necessary, if there is risk of death from pressure, and might be adopted more readily for the prevention of cerebral spastic paralysis. Cushing (1905) reported operations on 4 cases, one bilateral, with 2 recoveries. Lumbar puncture may reveal blood.

**Hæmorrhage into the Spinal Cord or Canal.**—This is commonly due to birth injury and on the ventral surface, perhaps spreading from the vertex. Small hæmorrhages into the cord are frequent and apparently unimportant; large ones are rare and possibly produce syringomyelia. The causes are the same as in intracranial hæmorrhage.

**Thrombosis.**—Primary thrombosis of cerebral sinuses or veins is due to marasmus, prolonged diarrhœa, broncho-pneumonia, tuberculosis, congenital syphilis, acute diseases, etc. Secondary or infective thrombosis is due to the streptococcus, pneumococcus, staphylococcus and other organisms; and occurs in general pyæmia, local abscess by extension through the wall, otitis media, cellulitis, mouth affections, etc. It gives rise to obstruction of the circulation, obliteration of the vessel, contraction of the clot, breaking down in the infective cases, pyæmia and meningitis.

*Symptoms* may be absent in the newborn, cachexia, and incomplete cases. Frequently they are only those of the antecedent disease. The ordinary cerebral signs are headache, restlessness, vomiting, delirium and coma. Infants may merely be somnolent or cry out at night. Vomiting, convulsions, coma and death occur in rapid sequence in some instances. Others show rigidity of the neck, trismus, squint, nystagmus, unequal pupils, irregular pulse and breathing. Optic neuritis may be present; and rigors and sweats in pyæmic cases.

In pure sinus thrombosis of the superior longitudinal, two lateral and cavernous sinuses, the symptoms are severe headache, vertigo, vomiting, dilated pupils, optic neuritis, and perhaps twitching of the face and neck muscles. It is indistinguishable from cerebral tumour.



Localising signs, often absent, are cyanosis and œdema of the forehead and dilated superficial scalp veins, if the clot is in the superior longitudinal sinus. If in the lateral sinus, the superficial veins on that side are empty; pain and stiffness extend into the internal jugular, perhaps causing dysphagia and lateral inclination of the head, and the vein may be palpable as a tender cord under the anterior edge of the sterno-mastoid; the deep cervical glands are enlarged; tenderness and œdema behind the mastoid result from thrombosis of the mastoid emissary vein; and bulbar symptoms may arise from pressure on nerves passing through the jugular foramen. Cerebral symptoms, fever and rigors indicate septic thrombosis of the lateral sinus, and the diagnosis depends on a thrombus in the jugular vein. When the cavernous sinus is blocked we find proptosis, œdema, amblyopia, swelling of the optic discs, retinal hæmorrhages, ocular palsy and panophthalmitis. Thrombosis of the ascending frontal, parietal and temporo-sphenoidal veins, may lead to acute red softening and hæmorrhage, terminating in sclerosis and palsy. Marasmic cases may live for weeks. A few primary ones recover by contraction of the clot. Secondary hydrocephalus may ensue if the sinus is obliterated. Pulmonary embolism may occur. Pyæmia is indicated by high fever, rigors, embolism and arthritis. Death is often due to multiple pyæmic abscesses in the lungs. Ligature of the vein, the sinus being incised and the clot removed, saves a few patients with thrombosis of the lateral sinus. It is usually due to ear disease.

*Embolism* is a sequel of endocarditis, or thrombosis of an artery in the brain or in a vein elsewhere. It causes red softening, an acute hæmorrhagic inflammation, followed by yellow softening from necrosis; occasionally white softening, from deficient blood supply. An infective embolus produces results closely allied to encephalitis. Both embolism and thrombosis are most frequent in the Sylvian artery and its branches. A large one causes convulsions, followed by coma, local paralytic and irritative symptoms, occasionally aphasia and sensory defects. The prodromata are such as are due to endarteritis or arterial thrombosis, viz. headache, restlessness, vomiting and drowsiness. It terminates in scar formation, sclerosis, cyst formation, and cerebral infantile palsy.

**Intracranial Tumours** are very uncommon under 2 years of age, except the pea-like tuberculous nodules sometimes present in tuberculous meningitis. After this age all kinds may be found, but in quite 50 per cent. the tumour is tuberculous, and 25 per cent. glioma or sarcoma. Other varieties are rare and secondary tumours almost unknown. The tuberculous tumours are often multiple, sharply limited, rarely break down, and may be surrounded by softening. They are most frequent in the cerebellum and pons. They are often latent, dense, have a greenish tinge, and end in meningitis. A local source of infection in the thoracic or mediastinal glands, occasionally in the skin, is almost always found. Gliomata are epithelial in origin, from the neuroglia cells, infiltrating in character, and liable to myxomatous



degeneration, softening and cystic formation, and hæmorrhage. They grow slowly and do not involve the bones of the skull or cause secondary deposits, and are most common in the pons. They are richly cellular, fibrillary, spider-celled, spindle-celled or giant-celled (Stroebe). Sarcomata are most common in the cerebellum. They are single, capable of removal, grow slowly, and are liable to hæmorrhage and myxomatous degeneration.

Cystic tumours, due to the cysticercus or echinococcus, are exceptional in this country but not uncommon in Europe. They may be single, form multiple cysts on the surface of the brain and in the ventricles, occur in the cranial bones, and are at times embolic, the result of dissemination by the blood during operation. Small ones may calcify. The progress is very slow and a few recover. Gummata are exceptional and are not due to congenital syphilis. Miliary aneurysms are due to chronic interstitial nephritis, infective emboli, or micro-organisms. Abscess receives separate consideration.

No part of the brain is exempt. About 30 per cent. of all tumours are cerebellar and 10 per cent. pontine. They may be cortical, cerebral, or basal. Supra-tentorial tumours are very rare under 15 and still more rare under 8 years of age, if tuberculous nodules are excepted.

Boys are twice as liable as girls, possibly because they are more likely to injure themselves. It is quite common for the tumour to correspond with the site of an injury, though it is difficult to believe that injury is an exciting cause.

*General Symptoms* depend on the size, rate of increase, the vascularity and nature of the growth. They are headache, vomiting, optic neuritis and atrophy, vertigo, bradycardia, and changes in disposition and mental activity. Yet headache, vomiting and optic neuritis may be absent in even large tumours. *Headache* is the most important sign. It varies in site, extent and severity. It is not as bad as in adults, severe at times, and constant or intermittent. It is exaggerated by mental effort and increased intracranial pressure. It varies from a dull, aching pain to paroxysmal agony in which death may occur. Marked variation may depend on the vascularity of the tumour. It is most valuable as a sign when it is persistent and localised. Occasionally it is over the site of the tumour, commonly frontal or occipital, rarely vertical or parietal. It is most constant and severe when the tumour is in the posterior cranial fossa, the pain being occipital and radiating into the neck. The pain may get less or disappear when hydrocephalus is apparent, because pressure is relieved. It is least constant and severe in infants for the skull bones can separate readily; and is difficult to diagnose in them, perhaps only giving rise to screaming attacks. A sense of fulness, pressure or constriction in the head may also be present. *Tenderness of the scalp* may be noted in cortical tumours, over a limited area and elicited by percussion. *Convulsions* are the next most frequent sign in children, still more so in infants. They may occur singly at long intervals or as often as 20-30 times a day. In cortical tumours they may be of the type of Jacksonian epilepsy. Often they are general and



may pass through the stages of an epileptic fit, ending in coma. Occasionally they leave behind a general fine tremor for hours or days. *Optic Neuritis and Atrophy* are sometimes the first sign, often present, almost always double, and independent of the size of the tumour. It is the type known as "choked disc." It appears in one eye first and is rarely of the same degree in both eyes. Limitation to one eye indicates disease in the optic nerve anterior to the chiasma; if in the chiasma, there is hemianopia also. It occurs in quite 75 per cent. and may be advanced though sight seems little impaired. Gradual loss of sight in children attracts little notice and the blindness may be thought of sudden onset. As the neuritis increases there is limitation of the field of vision for light or colour and finally amaurosis. It comes on earlier and is most intense in tumours of the cerebellum and basal ganglia, for intracranial pressure is most likely to be raised. True neuritis is due to distension of the nerve sheath, extension of irritation and inflammation along the nerve, or meningitis. *Vomiting* is more frequent than in adults and is cerebral in type. It may or may not be accompanied by nausea, may occur frequently and independently of food, is generally associated with optic neuritis and often with severe paroxysms of headache, sometimes with vertigo or on movement. It may be so bad as to cause inanition. It is rare in glioma of the pons, most common in sub-tentorial tumours, and not infrequently absent. Occasionally it simulates anorexia nervosa.

*Vertigo* is constant or paroxysmal with dizziness and sinking feeling in the stomach and mental confusion. The child feels as if falling or turning round (subjective), or as if the room and its contents are in motion (objective). It may come on while lying down, on change in position, or with vomiting. It is most common in tumours of the cerebellum or the posterior fossa of the skull. It must not be confused with the slight vertigo due to ocular palsies. *Nystagmus* may be present in late stages, and in hydrocephalus and cerebellar tumour. *Insomnia* is rare. The pulse is infrequent, slow and irregular; frequent and irregular towards the end. Bradycardia may be marked at the height of the headache. The pulse alters noticeably with change in position. Acceleration may depend on vagus palsy. Attacks of syncope are rare. Cheyne-Stokes breathing is often present in late stages. Fever results from secondary meningitis. The *psychical and mental changes* are first irritability, depression, disinclination for amusement and games, and a desire for quiet; sometimes outbreaks of irritability, screaming and apparent terror; or persistent somnolence and gradual cessation of speech. In older children there is dulness, stupidity, lack of interest and intelligence, and forgetfulness. Finally the child passes into coma, imbecility or dementia. In a few instances melancholia, hallucinations and maniacal attacks have been noted.

*Localising signs* may be few or absent, slight at first and increasing with the size of the tumour. A sudden increase is due to hæmorrhage into the growth or surrounding tissues. Some of the symptoms depend on local



destruction or irritation, others on interference with function by direct pressure, or on circulatory effects from pressure by disease in some other part of the brain. They are the same as in adults and include ataxia, imperfect gait, disorders of equilibrium, spasms, tremor, paralysis, altered reflexes, sensory disturbances, irregular flushing, polyuria, glycosuria, interference with swallowing, articulation and respiration, hallucinations of sight and hearing, affections of the special senses, hemianopia, disturbance of speech, and mental action and psychical changes.

The details of localisation must be studied in works on the nervous system. Cortical tumours may give rise to local headache, tenderness, and localised fits. One in the *frontal* lobe may cause dulness, apathy and inattention, fainting attacks, petit mal, drowsiness, enuresis and coma. If it extends backward it produces symptoms referable to pressure on the motor area, and convulsions on the opposite side of the body. Pressure on the olfactory lobe affects smell. Occasionally the growth may reach a large size and cause little mental deterioration. Motor aphasia and agraphia are due to backward pressure on the left side. A tumour in the *ascending frontal* lobe, or pressing on this lobe, causes motor symptoms and spasms. Attention must be directed to the point of origin of the subjective sensation and spasm, the order in which the spasm spreads, and the point of greatest subsequent paresis. Monoplegia is apt to result from a cortical, and hemiplegia from a sub-cortical growth. Sensory auræ, ushering in fits, are most common in tumours of the *ascending parietal* convolution. A growth in the angular gyrus may cause word-blindness and even temporary blindness of the opposite eye. Conjugate deviation of the eyes to the left is said by Wernicke to be due to a lesion in the left inferior parietal lobule. Fits of all kinds occur in tumours of the supra-marginal and angular gyrus, sometimes hemianopia on the opposite side, and impaired smell on the same side. Speech is unaffected in tumours on the right side.

Tumour in the *occipital* lobe causes bilateral homonymous hemianopia on the opposite side and sometimes partial loss of colour vision. If on the left side there may be word-blindness and psychical blindness, inability to recognise familiar faces and objects. Visual hallucinations may depend on cortical irritation of these lobes. Word-deafness is due to a tumour in the *temporo-sphenoidal* lobe. If in the *basal* ganglia, the signs are chiefly those of pressure on the internal capsule and on the cranial nerves outside the brain. A small abscess in the right corpus striatum of a 13-year old girl, due to infective endocarditis, caused rigidity of the limbs, chiefly on the left side, and left facial palsy. *Pontine and bulbar* tumours affect the motor and sensory tracts on the opposite side, and the fifth to the seventh cranial nerves on the same side. If in the upper part of the pons, the third and fifth nerves are affected; if in the lower part, the sixth, seventh and eighth; if in the medulla, the ninth to the twelfth nerves and the vasomotor centre are likely to be involved.



*Cerebellar Tumours* are outside and press on the cerebellum, in one lateral lobe, or in the vermiform process. Headache is severe, optic neuritis common, and vomiting nearly always present. Tumour is most frequent in the lateral lobe and can be successfully removed. If small, and not involving the median lobe, it may be latent or only cause general symptoms. If the middle lobe is involved, vertigo and ataxia are present. The vertigo is severe, fairly constant, and always in the same direction. It is exaggerated when the headache is severe and less marked between the paroxysms. It is usually subjective, the patient turning in the same direction if the tumour is intra-cerebellar, and in the opposite direction if it is extra-cerebellar. If objective, the objects move in a direction away from the tumour. There is often nausea, faintness and general bewilderment. The gait is staggering, as if intoxicated or on ship-board, the steps short and irregular, and the body swaying from side to side. In severe cases the patient cannot stand or even sit up in bed. If the tumour invades a cerebral peduncle, there is generally a tendency to fall toward the tumour. The ataxia is most in the leg on the side of the tumour, is always present in tumour of the middle lobe, and less with the eyes shut. In tumour of the vermes the tendency is to fall backward or forward. It is independent of vertigo, paralysis and loss of muscular sense. The seventh and eighth nerves are most often affected by extra-cerebellar tumours and tinnitus is present; in intra-cerebellar ones the fifth to the eighth may be involved. The knee jerks are variable, and the epigastric and plantar reflexes unaltered. In tumour of the vermes there may be fits in which there is retraction of the head and arching of the back. Occasional symptoms in late stages are tremor, paresis of the limbs and trunk muscles, ankleclonus, head-rolling, retraction of head, spastic rigidity, blindness, deafness, mental disturbance, convulsions and unconsciousness. Hydrocephalus is common. Cerebellar fits may be due to pressure on the pons and cerebellum, or irritation of the labyrinth. There is increased tinnitus, vertigo, blackness before the eyes, occasionally sudden blindness or unconsciousness, a tendency to fall, and extensor tonic spasm.

*Diagnosis.*—This depends on the general symptoms primarily and localising ones secondarily. Many of the later ones are due to circulatory disturbances and hydrocephalus. James Taylor bases a table, useful for localisation, on the presence of hemiplegia:—

- (1) Pure hemiplegia—the tumour above the tentorium.
- (2) With palsy of the opposite third nerve—in the crus cerebri.
- (3) With hemiataxy and nuclear ocular palsy—in the mesencephalon.
- (4) With hemiataxy on either side and palsy of the fifth, seventh and eighth nerves on the same side—in the pons Varolii.
- (5) Without facial palsy, with hemiataxy on the opposite side, and with alternate palsy of ninth to twelfth nerves—in the medulla.



Hemiataxy without hemiplegia indicates a sub-tentorial tumour. The diagnosis from abscess is considered under abscess. Meningitis may simulate and is more common than either, and is more often associated with abscess. Hydrocephalus may give rise to symptoms of tumour or may be secondary to it. Occasionally cerebral sclerosis may be suspected. Intracranial hæmorrhage produces well marked symptoms but they are not progressive.

*Prognosis.*—Tuberculous tumours may cease growing and undergo caseation. A similar result may ensue in gummata and possibly in gliomata. Shrivelled and atrophied tumours have been found after death. Recovery may leave behind permanent blindness. For this reason it is advisable to trephine if there is optic neuritis, for it may prevent blindness. Remarkable intermissions occur and all the symptoms, except blindness, disappear for a long time. Usually the course is progressive. The symptoms increase in intensity and others develop from extension of the growth, pressure and circulatory disturbance. Convulsions may be present throughout, especially in abscess, or only appear toward the end and as a terminal event. Death takes place from meningitis, suddenly from intense headache or bulbar palsy, hæmorrhage, or progressive coma and asthenia. Acute exacerbations are due to hæmorrhage, into the tumour or adjacent tissues, or surrounding softening. The average duration is less than two years.

**Cerebral Abscess.**—An acute abscess is generally traumatic and situated between the bone and dura mater (extra-dural), or between the dura mater and pia-arachnoid (sub-dural) and shut off. Multiple small pyæmic abscesses are a type of suppurative encephalitis. The common intracranial abscess is chronic, encephalic, and due to ear disease. Sometimes it is secondary to disease of the nose, orbit or cranial bones, and occasionally no cause is found. It may be secondary to infective endocarditis, empyema, bronchiectasis, pulmonary gangrene, thrush, tuberculosis and infective fevers, being generally multiple and pyæmic in these cases.

*Pathology.*—The inflammation starts in the white matter and ends in local suppuration. Pyæmic abscesses are multiple. The single abscess varies in size from a pea to that of a whole hemisphere. There is generally a definite limiting membrane, and the adjacent tissue is œdematous, softened or inflamed. Occasionally it becomes encapsuled, caseates, and remains latent until fresh inflammatory action leads to rapid increase in size, rupture into the ventricle or on to the surface, and suppurative meningitis. The contents vary from thick and greenish, perhaps inspissated and very offensive pus in chronic cases to thin, watery, brownish sero-pus in acute abscess, sometimes gas and pus. The capsule consists of thick fibrous tissue or necrotic brain substance.

*Symptoms and Course.*—It may be so acute as to defy diagnosis or so chronic as to simulate tumour. In its initial stage there may be no symptoms or a more or less acute period of 1-7 days illness with headache,



vomiting, prostration, variable fever and chilliness. This may be followed by drowsiness, vacancy, constipation, polyuria, glycosuria, coma, and death from respiratory failure or from meningitis. Or it may pass into a stage of complete latency; or incomplete latency in which the symptoms are variable headache, malaise, slight fever, chills, drowsiness, and perhaps convulsions, optic neuritis, irritability and mental changes. Occasionally the signs are acute and temporary, due to a local meningitis. The period of latency is rarely more than a few months but may last for years. As a sequence of this or of the initial stage an acute illness ensues, possibly due to suppurative cerebritis or meningitis. It is characterised by headache which may be localised, increased by cough and stooping; vomiting, especially in cerebellar abscess and on standing up; convulsions, loss of memory, stupor, delirium, and a normal or subnormal temperature. Bradycardia, arrhythmia, intermittence, tachycardia, slow breathing and occasionally Cheyne-Stokes respiration may be noted. In addition there may be the usual symptoms of a cerebral tumour and localising signs. Sometimes the symptoms are limited to persistent severe headache, dulness of intellect, subnormal temperature and infrequent pulse. Other cases may exhibit high fever, delirium, rigors, convulsions, etc., and end fatally in 7-10 days.

The abscess of ear disease is twice as common in the cerebrum as in the cerebellum, and more frequent under 10 years of age than in adults, in males, and on the right side. It is single in 90 per cent., usually in the temporo-sphenoidal lobe, occasionally in the lateral lobe of the cerebellum, and most liable to occur when the dura mater is implicated. The dura and brain between the diseased bone and the abscess are generally affected.

Extension of the mischief causes thrombosis of the lateral sinus, purulent meningitis by direct spread from the bone disease or rupture of the abscess, or rupture into the lateral ventricle, fourth ventricle or ear. An acute abscess is generally associated with sinus phlebitis and a chronic one with labyrinthine disease. A boy, 3 years old, had multiple fits for 2 months, somewhat staggering gait and tremor of the hands. After a few weeks' interval the fits recurred. The ataxia was chiefly on the left side. The left ear discharged pus at the end of 6 months. He became drowsy, aphasic and comatose, and died suddenly in a fit 12 months after the onset and 3 months after exploration of the brain. An abscess was found in the temporo-sphenoidal lobe after death. Fever was almost absent throughout.

*Diagnosis.*—In disease of the mastoid and middle ear the abscess is most likely to be temporo-sphenoidal; and there may be word-deafness and motor aphasia, if it is on the left side. Unilateral fits and hemiplegia are in favour of a parietal abscess. In disease of the labyrinth the abscess is more likely to be cerebellar and associated with rigidity of the neck, ataxia, vomiting and severe headache. Disorders of speech, hemiplegia and hemiparesis point to the cerebrum as the site of the mischief. In



differentiating abscess from tumour stress must be laid on the sudden onset of symptoms, which may subside and become latent; headache more often absent, optic neuritis less common, meningitis probable, course short, and mental changes rapid. In tumour the symptoms come on slowly, progress steadily, headache is more severe and paroxysmal, optic neuritis common, mental changes more gradual, the course prolonged, and hæmorrhage into the tumour is not infrequent. Yet a chronic abscess may be latent and the signs those of a tumour; and a tumour may be rapidly progressive, from hæmorrhage into its substance, or become obsolescent. In doubtful cases the diagnosis is based on a careful estimation which is the more probable. In some instances the signs are limited to those of atypical meningitis, or of pyæmia and marasmus with no definite brain symptoms. The diagnosis of chronic abscess from chronic tympanic disease, sinus thrombosis or meningitis is often impossible. The two latter affections may occur as complications and obscure the primary cause.

*Prognosis.*—Abscess may terminate in purulent meningitis from external rupture, rare except in cerebellar abscess, rupture into the ventricle, sudden and unexpected death, or death from respiratory failure during coma. Rupture into the ventricle causes severe symptoms and death from collapse in a few hours. In untreated cases the ordinary signs of a growing tumour develop. Optic neuritis is rarely followed by atrophy.

*Treatment of Tumour and Abscess.*—Many tumours being tuberculous and multiple it is advisable to adopt the treatment appropriate for tuberculosis. If there is the least possibility of cerebral syphilis mercury and pot. iod. must be given. Encapsulated tumours and cysts, especially in the frontal and parietal regions, are the most amenable to successful operation. Vascular infiltrating tumours are not removable. If measures, which increase intracranial pressure, increase the severity of the symptoms it is probable that the tumour is a vascular one. Trephining is advisable for choked disc and optic neuritis. As much as 7 dioptries of swelling have subsided and left perfect vision, but white exudation about the disc generally leads to some impairment of sight and atrophy. Lumbar puncture may relieve pressure. It is negative in abscess, unless there is meningitis. Both tumours and abscess are difficult to locate, to find at operation, and to remove. It is justifiable to explore the temporo-sphenoidal lobe, if there is evidence of intracranial abscess and the child is getting worse. Symptomatic treatment is directed to reducing intracranial pressure and cerebral congestion by rest, quiet, leeches behind the ears, mustard foot baths, calomel and saline purgatives, and vaso-dilators. Phenacetin and caffeine, bromide, chloral, morphia and other hypnotics may be needed for the relief of headache. Nasal feeding is often necessary. It was continued for 4 months in the case of a 6-year old boy with tuberculous tumour of the left lobe of the cerebellum and secondary hydrocephalus.



## CHAPTER LIII.

### POLIOENCEPHALITIS AND POLIOMYELITIS.

**Polioencephalitis.**—*Syn. : Acute Encephalitis — Cerebral Infantile Paralysis.*—This disease was first recognised by Strümpell in 1884. Since then evidence has accumulated in favour of the view that there is a specific disease of the nervous tissues, liable to occur in epidemic or family outbreaks, and apparently the result of the action of some toxin rather than of a specific micro-organism. No such organism has been found in the cerebro-spinal fluid or the nervous tissues. This suggests that the poison is not produced *in loco* but is carried to the brain and cord by the blood.

Acute encephalitis is due to many causes. It is apt to occur in specific fevers, notably measles, cerebrospinal and purulent meningitis, and as a complication or sequel of diphtheria, influenza, pneumonia, pertussis, vaccination and affections of the tonsils, and may follow bruising of the brain by traumatism. Septic infections in the newborn sometimes produce it, in the form of attacks difficult to distinguish from toxæmia. Predisposing causes are malformation of the brain, injury at birth, prematurity, cerebral syphilis and malnutrition. A localised distribution is the result of embolism in infective endocarditis, and thrombosis or hæmorrhage, as in pertussis and diphtheria ; possibly these causes only acting as predisposing ones and the actual disease being due to secondary toxæmia or infection. One-third of the hemiplegic cases follow acute specific fevers. An acute purulent encephalitis may be associated with purulent meningitis and due to infection.

The type under consideration is much more common. It is a primary specific disease of the grey matter of the central nervous system, which is divisible according to its distribution into the following groups. Many of the cases overlap and present symptoms of more than one group. All parts may be simultaneously affected.

#### I. *Polioencephalitis superior*, localised or general :—

- (1) Pre-frontal, causing profound mental changes.
- (2) Mid-cerebral, affecting the motor areas and producing hemiplegia or diplegia.
- (3) Occipital, perhaps causing blindness.
- (4) Temporo-sphenoidal, possibly setting up deaf-mutism.
- (5) Cerebellar, causing ataxia and disturbance of equilibrium.



II. *Polioencephalitis inferior* ; located in the pons or bulb and affecting the nuclei below the level of the corpora quadrigemina, producing palsy of one or more of the cranial nerves. In rare instances there is a polyneuritis of the cranial nerves and not an affection of the nuclei.

(1) Strabismus and varieties of ophthalmoplegia.

(2) Acute bulbar palsy, affecting one or more of the nerve nuclei.

III. *Diffuse disseminated encephalo-myelitis*, in which multiple inflammatory foci are distributed throughout the central nervous system. It simulates disseminated sclerosis but may end in recovery.

IV. *Poliomyelitis* or acute infantile paralysis.

V. *A neuritic type* in which the main stress falls on the peripheral nerves. It begins with pains in the limbs or joints and is followed by paralysis, with ultimately more or less complete recovery. It may be limited to cranial nerves.

*Pathology.*—The chief anatomical feature is the occurrence of minute hæmorrhages in the parts affected. The neuritic cases are explained as the result of irritation of the nerves or the nerve roots by extension from the pia-arachnoid of the brain or cord, which is believed to be always primarily affected. The post mortem signs are in favour of a toxic degeneration and opposed to inflammation, except in the cases which are distinguishable by the definite presence of purulent or cerebrospinal meningitis. Cantani produced it in rabbits by injecting the toxins of dead influenza bacilli. In many cerebral cases it might be spoken of as a general non-septic cerebritis, especially of the cortex in the motor area. The grey matter is reddish in colour ; the pia may be injected and adherent ; and on section the brain shows red or yellowish points surrounded by zones of œdema and softening. In later stages there is found degeneration of the nerve cells of the cortex and their nerve fibres, and descending degeneration of the motor tracts ; sometimes sclerosis and hydrocephalus. Similar changes are present in the cerebellum, if affected. The anatomical appearances vary with the duration of the illness. Both hyperæmia and softening are present ; and the brain may be soft, fluid, creamy, and contain many hæmorrhagic foci. A baby developed apparently sudden blindness. The pupils were small, equal and reacted slowly to light ; the optic discs were swollen temporarily. After death, which took place suddenly, the brain showed nothing except extreme softness. Other signs of infection or toxæmia, occasionally present, are myocardial degeneration, splenic enlargement, acute nephritis, and hæmorrhages into the intestines, mesentery and serous membranes.



Although the disease may occur at any age in the brain, quite two-thirds of the cases are under 3 years and it is rare after 7 years.

*Symptoms.*—The onset is usually sudden with headache, vomiting, pain in the back and limbs, and fever. The infant passes quickly into stupor, unconsciousness or coma; or may develop more or less paralysis without loss of consciousness. Cases differ much in their severity and may be characterised by convulsions, high fever and vomiting; fever and drowsiness, but no fits; apyrexia and screaming attacks; aphasia or almost sudden unconsciousness. Coma is rarely complete, the mental state being more one of hebetude or stupor. Convulsions are unilateral or general; single, multiple or continuous, and even fatal. In infants they are almost invariably present at the onset, and are unrelieved by ordinary measures. In mild cases the temperature is 101-102° F. and in severe ones 104-106° F. The pulse is feeble and frequent. The breathing is shallow and often of Cheyne-Stokes type; and attacks of asphyxia may occur. Irritability, restlessness and general hyperæsthesia are not rare. Squint, tense fontanelle, slight rigidity of the neck muscles, retraction of the head, and flexor rigidity of the limbs are more frequent. At the onset the reflexes are often absent if the cord is involved, and obtainable in cerebral cases. Sphincter control is usually lost.

The other symptoms depend on the extent and localisation of the mischief. New inflammatory areas may give rise to fresh convulsions. Unilateral ones (Jacksonian epilepsy) occur when the motor area on one side is attacked; and paralysis develops immediately and severely, or successively in the face, arm and leg. The loss of power is partial or complete; and the palsy is flaccid at first and spastic subsequently. It may be bilateral, if both motor regions are involved. There is no reaction of degeneration and no sensory disturbance. If the motor region escapes, the signs may be limited to affections of sight, hearing or speech. Occasionally there is optic neuritis and atrophy, aphasia or complete deafness. One child showed continuous tremor of the hands and forearms, without paralysis. Athetoid movements may be present.

The disease may chiefly affect the cranial nerve nuclei. A 4-year old boy was taken ill with fever, shivering and nausea. On the third day he vomited and had a fit or was delirious. Next day he became unconscious and lost sphincter control. He presented some rigidity of the neck muscles, trismus, conjugate deviation of the eyes to the left, and a temperature of 102·4° F. In the next 4 days he had 19 fits. The fever subsided in 24 hours. Consciousness returned on the eighth day of the illness. The conjugate deviation of the eyes persisted for 2 weeks; and he ultimately recovered with no ill effects except paresis of the muscles supplied by the right sixth and left seventh nerves. In bulbar cases the muscles of respiration and deglutition may be involved, and death ensues from bulbar palsy.



*Complications and Sequels.*—Some of these occur during the course of the disease and may be strictly regarded as symptoms. Chief among them are the motor defects, viz. hemiplegia, monoplegia, diplegia and crossed paralysis; clumsiness, fine or coarse tremor, choreiform movements, hemichorea, athetosis and ataxia, all of which are involuntary in some patients and in others only occur on voluntary movements; slow, stiff voluntary movements up to more or less rigidity; contractures and deformities; and various palsies of cranial nerves, chiefly the oculo-motor ones. The psychical disturbances are delirium, hallucinations, delusions, and screaming or apathy during the attacks; and subsequently weakness of memory and intellect, feeble-mindedness, or imbecility. Deaf-mutism, blindness, epilepsy and hysterical convulsions are occasional sequels. Epilepsy is general or Jacksonian. The child is not free from the liability to it until some years after puberty. The Jacksonian type usually appears in 1-10 years and is not severe. It is most likely to develop in children, over 2 years of age at the onset, for in them the mischief may be a focal hæmorrhagic encephalitis in the motor region, with red softening, yellow softening and sclerosis. Probably fits develop subsequently in about half these patients. *Focal encephalitis* at this age and later is milder in type than the acute encephalitis of infancy. It is more circumscribed, and more likely to end in recovery. The localising signs are unilateral tremor in one or both limbs, monoplegia or hemiplegia, facial palsy, aphasia, conjugate deviation of the eyes to the affected side, convergent squint and perhaps optic neuritis. It is this focal type of encephalitis which gives rise to cerebral spastic paralysis, and occasionally to spastic diplegia. It may end in cystic formation, porencephaly, abscess or sclerosis.

*Diagnosis.*—Encephalitis is liable to be mistaken for meningitis, poliomyelitis, sinus thrombosis, cerebral abscess or tumour, convulsions from other causes, and the onset of a specific fever. Serous apoplexy at first gives rise to similar symptoms but the intracranial pressure is generally much higher. In encephalitis it is by no means always raised, so lumbar puncture is of assistance in forming a conclusion. Frequently it is impossible to determine the exact nature of the illness until the course of the disease and the appearance of sequels afford further information. Paralysis, when present, is spastic in type.

*Prognosis.*—The course is variable. Impairment of consciousness may persist for a day or two, or for 1-3 weeks. The fever may be of short duration, or last for 2-3 weeks, rarely more than a week. The early symptoms indicate much more extensive mischief than there is evidence of subsequently. After a variable period the effects become more localised and it is possible to estimate the extent of the damage. Rarely the disease is subacute or chronic, and the effects appear slowly. Death may take place from cardiac or respiratory failure in a day or two, before accurate diagnosis is possible; or in 2-3 weeks from encephalitis, bulbar palsy, or



associated infective disease. Other patients recover gradually ; sometimes completely in 6-8 weeks ; more often with various sequels which may be permanent. Mental defect is quite common. Tremor, ataxia and nystagmus may persist for many months and yet disappear eventually.

*Treatment.*—Rest, bed, quiet, a dark room, and cold to the head are required. Calomel by mouth, warm foot baths, and leeches to the mastoids assist in cerebral depletion. Hydrotherapeutic measures are needed to reduce high fever. Feed carefully on light diet and allow no alcohol. Rely on chloral and bromides as nerve sedatives and for convulsions. Give pot. iod. if there is evidence of syphilis. Other measures are such as are suitable for meningitis. Lumbar puncture is advisable, if the fontanelle is tense and as an aid to diagnosis.

**Poliomyelitis.**—*Syn. : Acute Anterior Poliomyelitis—Spinal Infantile Paralysis — Infantile Paralysis — Infantile Atrophic Paralysis — Acute Atrophic Paralysis—Essential Paralysis of Children.* This disease may be defined as an acute specific infective fever of which poliomyelitis is a frequent, but not essential feature. It may occur without paralysis. If palsy is present it is the result of the anterior cornua being involved, and possibly this sometimes may be due to other causes. It is apparently analogous to encephalitis and similar in causation. The cord may be involved alone, or with the peripheral nerves, or the peripheral nerves only may be affected. In its purest form it is limited to the grey matter in the anterior cornua. It is sporadic and epidemic. The onset is more or less acute, usually with fever or gastro-enteric disturbance ; and palsy develops in a few hours or days. Some of the paralysed muscles undergo considerable atrophy, many recover, but it is rare for all the affected muscles to regain power completely. The usual result is a permanent paralysis and atrophy of one or more muscles or groups of muscles, and secondary deformities from unopposed contraction of their opponents.

The disease was described by Underwood of England in 1774. The first really good clinical account was published by Heine in 1860, he having observed it 20 years previously and suggested that the lesion was in the cord. The spinal lesion was demonstrated by Cornil in 1863, Prévost in 1865, and later and more fully by Charcot, Joffroy and Lockhart Clarke.

*Etiology.*—It has been ascribed to cold, wet, injury, teething, over-exertion and some general infection. Epidemics and sporadic cases have followed various specific fevers and other infective diseases. Isolated cases have been traced to sitting on wet grass, cold stones, or in a draught when heated. Over-exertion is possibly a predisposing cause by giving rise to fatigue of the motor cells. Some cases have followed falls and injuries. Perhaps injury to the back may set up neuritis, or myelitis from extension up the nerve roots. Dentition is a coincidence rather than a cause, except that it may predispose by giving rise to fever and disordered digestion. Most children are in perfect health at the onset. Some are enfeebled by



diarrhœa or wasting disease. Often the attack is ushered in by gastrointestinal symptoms ; or follows a specific fever or sore-throat. Accepting the infectious theory of origin, even the mildest sore-throat may prove the point of entry of the micro-organism. The disease is in no sense of the word hereditary, but a family disposition is sometimes noted. The occurrence of several cases in the same family is more likely to mean common exposure to infection than predisposition.

The youngest case on record is that of an infant in whom all the extremities were involved a few days after birth and extreme talipes equinus resulted (Kahler, 1888). Duchenne saw it in an infant aged 12 days. The youngest child under my notice was 6 weeks of age. It affects children of all ages from infancy to adolescence. Ninety per cent. of the cases occur during the first 5 years of life and most of these are infants under  $2\frac{1}{2}$  years. On account of its frequency during the period of dentition it has been called "teething paralysis." It is most common at 12-18 months. Boys are a little more susceptible than girls, and after the age of 10 years girls are very rarely affected.

Numerous epidemics are on record in America, Germany, Italy, Austria, Queensland, Norway and Sweden. From 1903-6 1,053 cases occurred in Norway in local epidemics. A big epidemic visited the United States, particularly New York, in 1908; about 3,000 cases. About three-quarters of the cases occur during the months of June to September, that is, in the hot time of the year when infection is rife.

Records of several cases in the same family are of considerable interest and illustrative of the association of this disease with encephalitis. The same association is better illustrated sometimes in the same patient. Pasteur (1897) recorded an outbreak affecting 7 children of one family within 3 weeks. All had moderate fever and headache. Three developed paralytic symptoms within a week ; in 2 fever was followed by tremors for a few days, with temporary squint in 1, but no paralysis ; in the remaining 2 no nervous disturbance was noted. Of the 3 paralytic cases 1 was infantile, 1 cerebral hemiplegic, and the other spastic spinal in type. Of 2 sisters one developed typical spinal palsy and the other cerebral hemiplegia (Sanger and Möbius). Cases have also been reported with spastic paralysis of one leg and atrophic paralysis of the other (Marie) ; right hemiplegia and aphasia with spasticity of the arm and subsequent atrophy of the leg (E. C. Williams) ; right hemiplegia and infantile palsy of the left leg (Neurath) ; spinal palsy and facial paralysis (Oppenheimer) ; acute poliomyelitis and ocular palsy ; and spastic hemiplegia, ocular palsy and infantile palsy of the opposite leg (Calabresse).

It may be urged in favour of the infectious hypothesis of origin that the disease frequently occurs in epidemics ; may affect more than one child in the same family ; occurs during or after an infective disease, and is most prevalent in hot weather. So far no specific organism has been implicated.



Possibly it may follow on various forms of infection. Schultze (1898) obtained the diplococcus intracellularis from the spinal fluid of a boy, aged 5 years, with acute infantile palsy and signs of meningitis. Geirsvold (Norway) also isolated a diplococcus from the cerebrospinal fluid before and after death. It was Gram-positive, produced short chains in broth, was pathogenic to mice and rabbits and frequently caused paralysis in them. He also noted that mild or abortive cases without palsy occurred during or before epidemics. They were associated with rigors, sore-throat, sweating, and sometimes signs of irritation of the central nervous system. In addition to typical paralytic cases he noted others of progressive, and sometimes fatal palsy, following convalescence.

Landsteiner and Popper (1909) transmitted the disease to monkeys by inoculating the peritoneal cavity with an emulsion of spinal cord from a fatal recent case. Landsteiner and Levaditi further transmitted it from one monkey to another, and ascertained that the virus could pass through a Berkefeld filter. Flexner and Lewis (1909-1910) confirmed these results and showed that, although the organism was invisible under the microscope, it would grow in cultures, best in human ascitic fluid bouillon. A monkey inoculated with such a 4-day old culture developed paralysis on the thirteenth day.

*Morbid Anatomy.*—The post mortem appearances vary with the date of examination after the onset of the attack. In a child, aged 5 years, who died a few hours after the onset, Drummond (1885) found the grey matter in the cornua unduly red, the capillaries distended and the vessels running from the surface to the cornua also distended, minute blood extravasations in the grey matter, swelling of the neuroglia cells, and nerve cells which were unduly granular. Examination made within 6 weeks of the onset has revealed foci of inflammatory softening in the anterior cornua, generally in the cervical and lumbar enlargements. These focal lesions do not usually extend beyond the neck of the cornua and are generally situated in the antero-internal and antero-external parts, even encroaching on the white matter on the external side. Sometimes small hæmorrhages and even actual cavities are present. The nerve cells have disappeared or are undergoing degeneration, with more or less disappearance of their processes. The inflammatory process is rarely limited to the foci but is usually widely spread, in slight degree, and may extend to the adjacent white columns. The posterior cornua are rarely affected. The blood vessels in the anterior horns are dilated, possibly thrombosed, and their walls are thickened and show nuclear proliferation.

After many years the affected part of the cord is often smaller than natural; perhaps the whole of one lateral half shows this diminution in size. On section the affected grey matter is more translucent than normal. The nerve cells may have totally gone or the few that remain are mere rounded, shrunken, deformed masses of protoplasm without nerve processes.



The nerve fibres have disappeared from the anterior horns and the neuroglia has increased. It consequently stains very deeply with carmine. The white matter and the nerve roots are smaller, and degeneration may be traced down the nerve to the muscles. The blood vessels in the cornua are thickened; the posterior horns are smaller, sometimes sclerosed; and there may be non-development or degeneration of Clarke's column.

The focal patches are usually unilateral, 5-25 cm. long, and may exist in the cord, bulb and pons. They may be multiple, sometimes bilateral, and are most frequent in the lumbar region. A consideration of these anatomical changes shows that in the first stage there is acute congestion of blood vessels and proliferation of the cellular elements of the adventitia. There is no evidence of ante-mortem thrombosis or changes in the intima. The perivascular lymph sheaths are crowded with round cells and there is an intense proliferation and cell infiltration round the blood vessels in those parts of the nervous system most freely supplied with blood. It is a diffuse inflammatory infiltration, and may be spoken of as an interstitial anterior poliomyelitis with subsequent degenerative changes in nerve cells. The atrophy of these cells follows more or less the distribution of the small arteries in the anterior horns, very suggestive of primary blood infection. More probably it is an acute perivascular inflammation of the pia-arachnoid which extends inwards, especially along the vessels in the anterior fissure. Cases have, however, been described in which the inflammation is apparently parenchymatous in type, an acute degeneration of the nerve cells which present granular degeneration although the interstitial tissue is unaffected. In old-standing cases there may be extremely slight interstitial change in contrast with the marked alteration in the nerve cells.

The muscles are greyish-red or yellowish-white in colour. Their fibres undergo granular degeneration and atrophy, with loss of transverse striation, and more or less completely disappear. The nuclei of their sheaths and of the interstitial tissue proliferate. Only a few fibres may be affected. They are small, and may be replaced by interstitial adipose or fibrous tissue. Those, in which granular degeneration is slight, may recover more or less completely. Some of the fibres may hypertrophy. The bones are smaller and thinner from non-development, and the whole limb smaller than a normal one; the blood vessels are truly atrophied.

*Pathology.*—The inflammatory lesion is due to some organism or toxin conveyed by the blood stream. The nerve cells suffer either from the direct action of the toxin or some inflammation of the supporting tissue. The changes in the cord are the effects, not the cause, of the constitutional state. Both are due to the same morbid process. The changes are located in the regions supplied by the anterior arteries of the cord and often limited to those supplied by the central system of spinal arteries. The anterior spinal artery gives off median branches which pass in at the anterior median fissure to each horn. These are terminal arteries, and hence infection is



purely local. There is no evidence that it is due to infective emboli or thrombosis. Probably it is primarily a vascular disease of the pia-arachnoid.

The theory of toxin causation is supported by the febrile onset, the distribution of the anatomical changes in the region supplied by the anterior arteries of the cord, the vascular changes, epidemicity, and the result of experiments on animals. The cerebrospinal fluid is clear and may contain an excess of polymorphs, but no organisms are found in it. Injections of toxins and various organisms into animals produce myelitis by direct action on the nerve parenchyma or on the vessel wall. Possibly these two processes are combined, or the toxins may set up acute degeneration of nerve cells.

The increased vascularity and functional activity of early life render these regions very liable to disease. The paralytic symptoms depend on the degeneration of the motor cells, which also leads to muscular atrophy and loss of reflexes. If the cells recover, their processes undergo regeneration and the muscles recover. The wide distribution of the initial paralysis is due to the wide distribution of the inflammatory mischief in the cord and perhaps secondary extension to the white columns. Subsequently the mischief becomes localised and its effects limited.

In cases of onset so sudden as to suggest focal hæmorrhage it is probable that there are mild inflammatory changes in the cord, not sufficiently severe to give rise to premonitory symptoms. Possibly a focal hæmorrhage results therefrom. It is unreasonable to suppose that local infective processes cannot take place in the cord without the antecedent condition of an acute constitutional state.

*Symptoms.*—The common type is associated with fever, vomiting and malaise for some hours or days before the onset of paralysis. In other cases the onset is sudden, with no previous sign of ill-health, pain or alteration in sensation. These latter cases are most frequent during the first 2 years of life and the paralysis is usually limited to 1 limb. In epidemics there are numerous aberrant types.

The disease may be divided into the initial stage; the paralytic stage, primary and permanent; and the stage of atrophy and contractures. Or one may speak of an onset lasting 1-7 days; a period of quiescence or fixation of 1-4 weeks; partial recovery, 1-6 months; and a subsequent chronic condition. The incubation period is unknown.

The onset is very variable and the affection frequently overlooked in the early stages. In many cases the child is taken to the doctor because the affected limb is always cold and does not grow as well as the other. There may be no history obtainable as to when the loss of power first began. Sometimes the child goes to bed apparently well, may be a little restless during the night, and awakes with one or more limbs paralysed. This was called by West "paralysis in the morning." Or the palsy may come on quite suddenly like that of spinal hæmorrhage. A few cases occur during



the course of an acute fever or during convalescence, the paralysis being the first symptom to attract attention. Others are preceded by pain in the limbs, commonly ascribed to rheumatism or "growing pains."

The usual mode of onset is a febrile attack of variable severity with a temperature of 101-105° F. It is generally accompanied by gastro-enteric disturbance, as a cause or consequence of the fever. Vomiting is a frequent early symptom, and diarrhoea not uncommon; sometimes there is constipation. Anorexia and general malaise are present. In bad cases the fever is associated with severe nervous disturbance; headache, restlessness, insomnia, twitchings, convulsions, delirium, or somnolence passing into coma and ending fatally. Head retraction, rigidity, and pains in the head and neck, back and limbs, may lead to erroneous diagnosis of cerebrospinal meningitis. The severe limb pains are due to involvement of the nerve roots. Sometimes they are the chief symptom (neuritic type). Screaming and crying may be present, and occasionally general immobility. On account of the vomiting the attack is often ascribed to simple stomach disturbance and its true nature is not realised until paralysis is evident, especially if the constitutional disturbance is severe. Loss of power is apt to be ascribed to the weakness and inertia of illness. Occasionally the constitutional disturbance follows the onset of paralysis. The fever varies in severity and duration. It usually lasts for a few days to a week, rarely for 3 weeks, and gradually subsides.

The *paralysis* comes on and reaches its maximum in 12-24 hours, or may be the first symptom; occasionally it takes 2 or 3 weeks to develop fully. It may begin in 1 limb and spread to the others in a few hours or days; but it is quite exceptional for extension to occur as long as 2 weeks after the onset. Extremely sudden onset may be due to hæmorrhage and, if this is in the cervical region, paralysis may be universal. Cases of this type may possibly be due to other causes, but their effects on the muscles are the same. The lumbar, cervical, dorsal and bulbar regions are affected in order of frequency.

All varieties of paralysis are produced. The left side of the body seems rather more liable than the right, and the left leg most of all. The leg muscles are more often affected than those of the thigh. One or both legs are affected in quite two-thirds of the cases. In order of frequency the paralysis involves 1 lower limb, both lower limbs, all the limbs with the neck and back muscles, 1 upper limb, 1 arm and 1 leg on the same side, 3 limbs, an arm and leg on opposite sides, both upper limbs. The paralysis may be limited to 1 muscle, or a group of muscles, usually functionally associated. Thus, a tibialis anticus may be affected alone, or in conjunction with the peronei and the extensors of the toes. The order of frequency in the various muscles of the legs is the peronei, tibiales, extensors of the toes, quadriceps, ham-strings and muscles of the calf, while the ilio-psoas generally escapes. Occasionally the psoas, iliacus, gluteus and thigh muscles are all involved, but the sartorius is spared. In the upper



extremities the deltoid; deltoid, supraspinatus and infraspinatus; the biceps and supinators; and the flexors of the hand, is the usual order of frequency. The clavicular part of the deltoid and serratus magnus muscle; the middle and posterior parts of the deltoid with the infraspinatus and rhomboids; the deltoid and the intrinsic muscles of the hand; the serratus magnus and pectoralis major; the middle portion of the trapezius with the other scapular muscles; are combinations of muscles which may be simultaneously affected. The diaphragm and the muscles of the abdomen, chest and neck are less often affected.

Unilateral paralysis of abdominal muscles leads to "*ballooning*." Paralysis of the erector spinæ is a serious cause of lateral curvature. The muscles supplied by the cranial nerves and the muscles of swallowing are affected, if the bulbar and pontine region is involved.

The sphincters generally escape, unless the centres in the lumbar cord are involved or the attack is very severe. Incontinence occurs in about 1 per cent. and retention in 5 per cent., but is generally dependent on the severity of the fever or on stupor. Sphincter trouble is not permanent.

Initial loss of power is greater than permanent paralysis, and rarely passes completely away. Improvement begins in a few days though little change is noticeable for 2 or 3 weeks. This is the stage of regression, fixation or quiescence. The parts first affected are the last to show improvement. There may be no definite signs of improvement for some weeks. Usually in 3 months those muscles, which will not be permanently affected, recover almost completely. In the others distinct wasting commences in 2-3 weeks and steadily progresses. In very fat children it may not be evident to the eye, but the muscles feel flabby and measurement shows decrease in size. Atrophy persists when it is once developed, though there may be considerable recovery due to hypertrophy of unaffected muscle fibres.

The affection may end in complete or partial recovery or complete paralysis. In partial recovery some of the muscle fibres remain paralysed, eventually atrophy, and are replaced by fibrous tissue. The tone of the muscle is diminished and it becomes stretched by the action of its opponents. This is still more marked in complete paralysis for the muscle fibres are replaced by fibrous and fatty tissue. The opponent muscles are physiologically shrunk by over-contraction, producing a deformity which can be easily overcome. If neglected it ends in pathological shrinking and a certain amount of fibrosis, with contractures and permanent deformity. Eventually fascia, ligaments and even bones become involved.

*Electrical Examination.*—Loss of faradic irritability may be evident as early as the fifth day. The muscles in which the reaction is diminished, and not abolished, recover. When atrophy is complete, loss of faradic irritability is permanent. Muscles which react at the end of 2 weeks may recover in a fairly short time. Faradic irritability usually returns in 6-12 months, on recovery of some of the muscle fibres. Galvanic irritability is



increased at first in the muscles in which paralysis will persist, but it slowly diminishes as atrophy continues and is lost in a year or two. If the muscles are persistently treated by electrical methods, the reaction to galvanism may persist for years although there is no return of power. The reaction of degeneration is present in the atrophied muscles. Idio-muscular contractility may be increased in the muscles which will be permanently affected, and subsequently disappears. The motor nerves degenerate and lose faradic irritability.

*Reflexes.*—The skin reflex is lost with the loss of muscular power and returns if recovery takes place. The knee jerk is lost in paraplegic cases and if the vastus internus of the knee is even slightly affected. The Tendo-Achilles reflex is lost if the calf muscles are paralysed. Exaggeration of reflexes may be due to slight degeneration of the motor tract, from an extension of inflammatory processes. It is most likely to be found in severe affections of the cervical portion of the cord or sudden hæmorrhage. The lower limbs are then completely paralysed but soon recover.

*Sensory Effects* are due to the inflammatory processes involving the sensory tracts in the cord, the nerves or nerve sheaths, or to spinal meningitis. In the last event marked rigidity of one or more limbs is associated with pain in early stages, and induced by movement. Pain is not infrequently felt in the nerves, muscles, or limb as a whole. It is most common in the back. Sometimes there is pain in the joints, the back and the neck and occasionally the pain in the neck is associated with slight rigidity. There may be marked tenderness in addition. Tingling, pins-and-needles and formication have been noted in patients old enough to describe their sensations. Anæsthesia has occurred in severe involvement of the lumbar cord, persisting for some weeks and associated with incontinence of urine.

*Trophic Effects* develop later. The growth of the limb is retarded or completely arrested. The younger the child at the onset of the attack and the greater the severity, the more marked is the subsequent difference between the 2 limbs. The skin is thin and liable to excoriations, ulcers and chilblains. It is colder than natural, mottled like marble, and becomes red and blue in cold weather. Hypertrophy of the hair and excessive secretion of sweat have been noted. Considerable adipose tissue may be deposited in the wasted muscles, (the local obesity of Landouzy), and cause a pseudo-hypertrophy. The bones are thinned and readily fracture. The joints are liable to partial or complete dislocation, from relaxation of the ligaments and lack of support by the tendons of paralysed muscles. The movements of such a joint are flaccid and flail-like.

*Deformities* are many and varied and depend on the unopposed action of the unaffected or less affected muscles, which contract and become permanently shortened. Partly they depend on extraneous causes. The weight of the bed clothes assists in the production of talipes equinus; and



shortening of a limb causes lateral curvature. The common deformities are those dependent on the common distribution of paralysis. The extensors of the thigh are much more often affected than the flexors. Talipes equinus is the most frequent. T. valgus due to the paralysis of the tibialis anticus, and T. varus due to paralysis of the peronei, are both associated with a certain amount of equinus. T. calcaneus, due to paralysis of the calf muscles, is rare. Pes planus is due to paralysis of the peronei and plantar flexors. In the upper arm the type of paralysis may be that known as Erb's paralysis or the "upper arm type," but usually the triceps is also involved. Club-hand is not uncommon. Paralysis of the serratus magnus produces the "winged scapula." Lateral curvature of the spine, unilateral distension of the abdomen, and other deformities also occur.

*Diagnosis* depends on the paralysis, atrophy, loss of reflex action, electrical reactions, and absence of sensory disturbance. The early febrile stage may be ascribed to other febrile causes or to disorder of the stomach. Paralysis may be mistaken for weakness or prostration, although prostration does not cause complete immobility. Cases in which there are sensory disturbances, pains in the course of the nerves in the paralysed limbs or in the back, and subjective sensations such as formication and tingling, may be put down to rheumatism, spinal meningitis or peripheral neuritis. In generalised peripheral neuritis the pain along the course of the nerves is very definite, the attack is more gradual in onset, and it can be traced to an antecedent diphtheria, influenza, arsenic, etc.; and the paralysis clears up. If however the acute symptoms have subsided, the presence of paralysis, atrophy, and electrical reactions similar to those of acute poliomyelitis, renders a differential diagnosis impossible. The wide distribution, even at the end of some weeks, and subsequent recovery are in favour of multiple neuritis. Some of these cases are instances of the neuritic type of the disease. The paraplegia is readily distinguished from that of transverse myelitis in which are found exaggerated knee jerks, anæsthesia, sphincter troubles, tendency to bed sores, no reaction of degeneration, and only slight wasting. Transverse myelitis is usually dorsal and due to spinal caries. Cerebral palsy is distinguished by rigidity, exaggerated reflexes and no reaction of degeneration. Paralytic chorea of a severe type creates considerable difficulty, unless the muscles are examined electrically. There is usually a history of chorea and evidence of some choreic movement on examination, and possibly some cardiac affection. Limitation of palsy to 1 shoulder in early life may be mistaken for a birth paralysis of the upper arm type, but the infraspinatus escapes and there is no inward rotation of the arm.

The disease has to be differentiated from the pseudo-paralysis of scurvy and congenital syphilis, the extreme paraplegic debility of rickets, and the delayed development of the lower limbs and of walking in backward and imbecile children. Other affections to be borne in mind are progressive muscular atrophy, spinal muscular atrophy (Werdnig-Hoffmann), Tooth's



peroneal type, muscular dystrophies, myatonia, spina bifida occulta, hæmorrhage into the spinal cord and hysteria.

*Prognosis.*—The disease is rarely fatal, even in its most acute stages, though it may end in death if the bulb is involved. Possibly some deaths from acute fever, before the paralysis has developed and made the diagnosis evident, are due to this disease. If the respiratory muscles are involved death may ensue from bronchitis or pulmonary catarrh. Complete recovery is possible but rare. In the “transient form of paralysis” the effects clear up in a few days. Almost always some permanent palsy and atrophy remain. In epidemics death and complete recovery are more common.

Even in the worst cases the relatives may be assured that considerable recovery will take place. The outlook depends largely upon the results of electrical examination, made when there is no progressive palsy, that is 7-14 days after the onset. Loss of faradic irritability soon after the onset or during the first fortnight indicates that the muscles will waste rapidly and probably remain permanently paralysed; the earlier it is lost, the worse is the prognosis. If the response to faradism is normal, the muscles will recover in 4-6 weeks. If there is a fairly good response at the end of 10 days and somewhat more diminished response at the end of 2 or 3 weeks, there is a fair chance of recovery. The return of faradic irritability is a sign of improvement and that considerable recovery will take place. If it is absent for 6 months the muscles are unlikely to recover and after 12 months practically certain not to, for it indicates persistent nerve degeneration. If there is response to faradism at the end of 2 years, there is still possibility of further improvement by electrical treatment. If there is no response to either faradism or galvanism after 2 years, the chance of improvement is very small. The presence of galvanic irritability shows that the muscle fibres have not undergone destructive degeneration, but if, after 3 months, there is no return of voluntary power in these muscles and faradic irritability is still absent, there will be no recovery. As long as the reaction of degeneration is present there is hope of improvement for the cells in the anterior cornua are not completely destroyed.

Generally speaking, more or less recovery takes place for 2 years and then the residual paralysis must be regarded as permanent. Late improvement is merely one which has been delayed by the too early use of mechanical appliances, or is due to volitional adaptations in the process of growth and development. The apparent exaggeration of the paralytic effects is due to the arrested growth of the affected limb. Progressive atrophy, starting in the affected limb or the parts adjoining, is a rare sequel. It is quite exceptional for any sequelæ to occur beyond the mechanical effects, due to the paralysis, and the trophic effects due to the deficient nerve and blood supply. The disease does not diminish the expectation of life.

*Treatment.*—During the acute stage the child is kept in bed in the prone or lateral position on light diet, and the fever treated on general principles



by diaphoretics, diuretics and mild purgatives. It is doubtful whether hydrotherapeutic measures for the reduction of fever have any effect on the process in the cord. Local applications of poultices, fomentations, ice bags, dry cupping, mustard paste and Pacquelin's cautery may be tried. Salicylates, aspirin, ergot and belladonna have all been recommended, and opium for severe pain. It is impossible to be certain that the treatment in any particular case has any definite effect on the course of the disease. The limbs must be kept dry and warm; and belladonna and glycerine may be applied for the relief of pain.

After the acute stage give drugs which improve the general nutrition. In 3-4 weeks strychnia or nux vomica can be used. Drugs to maintain or improve the general nutrition are just as beneficial. As soon as the acute symptoms have subsided or after the lapse of 2-4 weeks, massage and electrical treatment may be commenced. It maintains and improves the nutrition of the muscles during the time the nerve elements are recovering. A weak faradic current may be employed for those muscles which react to it. Some say that the faradic current increases the tendency to atrophy. Lewis Jones advocates the use of the sinusoidal current from the mains in electric baths. Galvanism is more readily applicable. Use 2 flat electrodes, applying the kathode to the affected limb and the anode to the spine. Begin with a current just sufficient to cause contraction, or none at all for a day or two if the child is frightened. At first apply it on alternate days and then daily for 10 minutes at a time for a few months, using weak currents. Continue it 2 or 3 times a week, as long as there is any improvement or for at least a year. The galvanic bath is most suitable for paraplegic cases. Friction and massage must be carried on systematically for months, to maintain nutrition and prevent deformity. Begin passive and active movements of the affected muscles as soon as the palsy is quiescent. The child at this stage is kept on ordinary diet. The affected limbs must be warmly clad and a warm bottle used if necessary. Hot salt baths do no harm. From the onset a cradle must be used to prevent T. equinus.

The treatment may be summed up in a few lines. Maintain the nutrition of the muscles. Keep the limb in a normal position, for stretched paralysed muscles recover badly. Prevent contractures and stretching of muscles, tendons and ligaments. Use simple malleable splints as supports by night and day. Continue electrical treatment for 6-12 months, massage for 1-2 years, and then rely upon growth and systematic education of the muscles. For this it is not necessary to send the child to a specialist or a foreign professor of gymnastics. The ordinary medical attendant can devise suitable exercises and teach the nurse to carry them out.

Mechanical treatment helps to prevent deformity and overcome deformity already present. If the lower limb is affected, keep it in the position of slight inward rotation of the hip, with the knee extended by sand bags; and apply a tin splint to the foot, keeping the ankle at right



angles. If the deltoid is affected, support the weight of the arm and keep it at a right angle with the trunk, to prevent subluxation of the humerus. A support will enable the child to sit up when, from weakness of the spinal muscles, he can not. It will enable the child to walk at an earlier period and thus keep in action the muscles which would otherwise remain idle. No support must be constantly worn, for under such circumstances the muscles become still feebler from disuse, instead of increasing in strength. All mechanical treatment must be supplemented by exercises to develop the affected muscles which are weakened by palsy, disuse and stretching.

Surgical measures after the lapse of 6 months may be required to correct deformity and enable the child to walk better. Many muscles are supposed to be paralysed though merely powerless from disuse. One group of muscles may be placed at a mechanical disadvantage and become useless because an opposing group has recovered first and contracted. Tenotomy often enables the child to plant the foot flat and firmly on the ground. Both simple section and shortening of the tendon are often of only temporary benefit, because the unaffected muscles still act too strongly. Transplantation of tendon or muscle, or the splitting of the tendon of a healthy muscle and attaching a portion of it to the tendon of a paralysed opponent (*muscle grafting*) are often of more benefit. The artificial production of ankylosis (*arthrodesis*) in flail joints is beneficial, but should not be done under 10 years of age or possibly not until the patient is old enough to decide whether he would prefer an apparatus to a stiff joint. In the case of a flexed wrist considerable improvement may result from cutting the flexors and placing the wrist in a position of hyper-extension for many months, to allow contraction of the stretched extensors, provided that on further flexion the patient can slightly extend the fingers. Nerve anastomosis may do good, if done 4-6 months after the onset.

Unfortunately in many cases all treatment proves unavailing to completely restore the damaged functions and the best that can be hoped for is a more or less imperfect cure. Severe deformities in simple cases are generally the result of insufficient care in directing the treatment to the prevention of their occurrence.



## CHAPTER LIV.

### THE SPINAL CORD AND THE NERVES.

*Myelitis—Friedreich's Disease—Tabes Dorsalis—Insular Sclerosis—Paralysis Agitans—Tumours—Birth Palsy—Pressure Paralysis—Peripheral and Cranial Nerves—The Sympathetic Nervous System.*

**Myelitis** is an occasional sequel of measles, typhoid fever and other infections. It commonly attacks both the cervical and lumbar regions ; and gives rise to paraplegia, exaggerated reflexes, sphincter and sensory troubles, and bed-sores.

**Pressure Myelitis.**—*Syn. : Pressure or Spastic Paraplegia—Spinal Caries—Pott's Disease—Angular Curvature.*—Apart from a cerebral origin, the common cause of spastic paraplegia in children is the pressure secondary to tuberculous caries of the vertebral column, which produces angular curvature of the spine. The process starts in the body of the vertebra, attacks several, causes destruction of those affected, and ends in approximation of the unaffected discs. The cartilages are little or not at all involved. Inflammation spreads to the membranes, and produces so much granulation tissue and thickening that the cord is compressed, and a mild form of myelitis is set up. Œdema and softening are followed by sclerosis and descending degeneration of the motor tracts and some degeneration of the ascending tracts. The nerve roots are often involved and give rise to sensory and motor symptoms, little noticeable if the caries is dorsal. Marked angular curvature may exist without signs of pressure, but the cord and nerves can be compressed by the falling together of the vertebral bodies. Abscess and bony sequestra pressing on the cord are rare. A posterior abscess is more common than an anterior one, and both may co-exist. The etiology is that of tuberculosis. Sometimes there is a history of a fall or injury. It may be present at birth or any subsequent age.

The *symptoms* vary with the site of the disease. It is most common in the dorsal or dorso-lumbar region, less frequent in the cervical, and least common in the lumbar. Either pain or paralysis may be the first sign. In the early stages of dorsal caries there are vague pains in the back and disinclination or inability to play games. The child is easily tired in the legs and disinclined to use them, sometimes lame, and may suffer from starting pains and nocturnal enuresis. Or there may be stumbling, due to



catching the toes, dragging the feet, and a girdle pain which may be described as stomach-ache. Gradually the weakness increases and the gait becomes stooping, with the back held rigid.

On examination we find rigidity of the spinal column, and inability to flex, extend and bend it fully in various directions without discomfort or actual pain; and perhaps pain on applying sharp, sudden pressure to the head or shoulders, or on lateral pressure on the spine in the rigid area. Sometimes there is local tenderness. The legs show rigidity, exaggerated reflexes, weakness and paraplegia. Ankleclonus and an extensor plantar reflex are often present. Spastic paraplegia is progressive, with impairment or lack of sphincter control, girdle pains, and slight or no sensory disturbance. Bed-sores appear. The angular curve is not present in early stages, and is found more often in dorsal than in cervical or lumbar caries. It develops gradually, and varies in extent and its effects with the site and extent of the disease.

Cervical caries is characterised by a striking manner of holding and moving the head. It is moved as a whole with the shoulders, and held as if the child was carrying a burden on the top and was afraid of dropping it. There is much thickening and infiltration of the tissues adjacent to the spine, little or no curvature, occasionally marked kyphosis, great rigidity, muscular spasm and sometimes torticollis. There may be loss of power in the thoracic and abdominal muscles, involvement of the phrenics and, if the disease is in the first and second vertebræ, bulbar symptoms and sudden death. The upper limbs are affected as well as the lower ones. Occasionally it is unilateral in its effects, and there is often some wasting of the small muscles of the hands. Numbness and weakness are early signs. The sphincters frequently escape.

In lumbar caries the rigidity of the spinal column is marked, lordosis pronounced, the muscles waste, the knee jerks may be lost, and there is greater liability to bed-sores.

*Complications.*—Psoas abscess is due to direct infection of the muscle by extension from the vertebra, generally in the lumbar but occasionally tracking down from the dorsal region. The abscess eventually points below Poupart's ligament and external to the main vessels, above this ligament, or behind in Petit's triangle.

*Diagnosis.*—Kyphosis is conclusive proof that spastic paraplegia is due to angular curvature, provided that the kyphosis from general convex curvature of the spine in severe rickets is excluded. Rigidity occurs in other diseases. X-rays may be of assistance. Psoas abscess may be the only symptom.

The *prognosis* is fairly good if suitable and prolonged treatment can be carried out. It depends on the general health. About half the patients die. Paralysis is rarely complete and usually subsides more or less.



completely after 3-9 months treatment. Death may be due to general tuberculosis or lung disease. Meningitis is comparatively rare.

*Treatment.*—If Pott's disease is properly treated from the onset, paraplegia will not develop. The chief measures are seaside and sanatorium treatment; good hygiene, food and nursing; cod-liver oil and syr. fer. iod.; and complete rest in the recumbent posture with extension and counter-extension, if necessary, and fixation of the spine by sand bags, poroplastic splints and such-like measures. Rigid supports must be continued for months after the pressure symptoms have subsided.

Operation is indicated in acute cases which involve sensory nerves, the bladder and rectum; if prolonged expectant treatment fails; and in cervical caries, if there is pressure on the phrenics and interference with breathing. It is contra-indicated in the presence of tuberculous disease elsewhere, if mechanical treatment has not been well tried, and if there are insufficient means for proper operation, nursing and subsequent treatment. Except in those cases in which granulation tissue or a caseous mass presses on the cord, operation is unnecessary and recovery will ensue equally well or better under simpler measures. Chipault collected 15 recoveries out of 103 cases treated by laminectomy. It must be followed by prolonged rest and sanatorium treatment. If the bodies of 2 or more vertebræ are destroyed and the laminae are then removed, there is no support left for the spinal cord. The less sensation is affected the better is the prognosis, whether the case is treated by rest or by operation.

A psoas abscess is treated by aspiration if the wound cannot be kept aseptic. Otherwise it should be evacuated. Make a small incision where the abscess points, irrigate with normal saline until the fluid returns clear, and then sew up the wound. The injection of iodoform emulsion is liable to set up toxic symptoms.

**Friedreich's Disease.**—*Syn.: Hereditary Ataxia—Hereditary Tabes.*—There are three varieties of hereditary ataxia. In the cerebellar ataxia of Marie and Sanger Brown the cerebellar symptoms predominate and the disease begins in later life (p. 683). In Friedreich's disease the spinal symptoms are in excess. In a third type there are additional muscular changes. Intermediate varieties exist. Friedreich's ataxia was described by him in 1863 as a form of locomotor ataxia. It is a familial endogenous affection of the spinal cord, of very gradual development, a slow and progressive ataxia. In about one-fourth of over 200 recorded cases there was some form of ataxia in one of the parents. No definite etiological factor has been found. The influence of acute febrile disease in precipitating an attack has been frequently noted. Several children in a family may be affected, usually at about the same age and of the same sex. Isolated cases are more likely to occur in small families. About one-fourth of the cases begin before the commencement of the second dentition, a few in infancy, and three-fourths before puberty. It rarely begins after 16 years of age.



*Symptoms.*—The first signs are weakness and unsteadiness in the muscles of the lower extremities, perhaps a difficulty in getting upstairs because of weakness of the leg muscles. Rarely it begins in the arms or with bulbar symptoms. The symptoms are motor, sensory, reflex, ocular, cerebral, genito-urinary, trophic and vasomotor. First in importance comes the disorder of *gait*. It is usually described as oscillatory, reeling (*cerebellar reel*), or like that of a drunken man, with lateral projection of the feet instead of the forward tendency seen in tabes. The legs are wide apart and the steps irregular and awkward, but not so violent, so sudden or so unreasonable as in tabes. The toes may be in-turned. The ataxia is often more marked on closing the eyes. Static ataxia is present in about half. Romberg's symptom is often present at the same time but is less frequent in the spinal than the cerebellar type. Inco-ordination may be so marked as to render the child unable to walk without the aid of crutches. In a few years ataxia is present in the upper limbs on movement and is at times increased by closing the eyes. In prehension the hand is spread out like a claw, from weakness of the interossei. Similar claw-like movements may occur in the foot, from hyper-extension of the toes. Retraction of the big toe is an early sign.

Tremor, like that of insular sclerosis and probably due to static ataxia, is not often present. Abrupt, choreiform movements, rather like those of chorea, may occur in the limbs, face and neck. Spasmodic contraction of the lower limbs, while sitting or lying, begins early. Later signs are permanent contractures such as talipes cavus, equinus and equino-varus, and scoliosis. Talipes cavus with full extension of the toes is most typical. The deformity may be the cause of the child coming under observation. Apparent palsy in early stages is due to inco-ordination. True loss of power, especially in the flexors of the leg and the spinal muscles, is of later development. The reaction of degeneration is rarely present.

Muscular sense is unaffected. Anæsthesia and analgesia seldom occur. Cutaneous sensibility is sometimes diminished, rarely increased. Slight pain is not infrequent in the early stages. Paræsthesiæ and girdle sensation are rare.

The *reflexes* vary with the predominance of cerebellar or spinal symptoms, and may be different in the two legs. In the spinal type the knee jerk is lost early. In the cerebellar type it is diminished, normal or even exaggerated. Other deep reflexes show similar changes. Babinski's sign is present. The cutaneous reflexes diminish as the disease progresses. Muscular atrophy is due to disuse and most marked in the lower limbs. There may be peroneal or other types of atrophy.

Disorder of *speech* depends on motor disturbance of the muscles of articulation and is most common in the spinal type. Speech is slow, awkward, jerky, stuttering, scanning, or moderately rapid and interrupted by sudden irregular pauses. Pronunciation is indistinct. Some-



times speech is unaffected. Fibrillary tremor of the tongue is not uncommon. Mastication and deglutition are rarely affected.

The expression is vacant and apathetic. The pupils react normally. Nystagmus is common in the spinal and absent in the cerebellar type. In the latter there may be squint, ptosis, diplopia and optic atrophy. Vertigo may be constant or occur in paroxysms. Later and rarer symptoms are headache, migraine, enuresis, disorders of the bladder and rectum, delayed sexual instinct and menstruation, impotence, palpitations, tachycardia, salivation, profuse sweating, dyspnœa and gastric symptoms. Some of these are of bulbar origin. At first the intellect is unimpaired; later it is weak relatively and there may be dementia. There is much emotional instability and proneness to laughter. Œdema and trophic ulcers of the legs have been noted in some prolonged cases (A. R. Moody, 1910).

*Morbid Anatomy.*—The cord is reduced to two-thirds the normal size in the dorsal region. The posterior root zones are degenerated and the columns of Goll, from the lower end to the tip of the calamus scriptorius, are sclerosed. Burdach's columns are less affected. The direct cerebellar tract is sclerosed throughout, especially in the upper dorsal region, and the column of Clarke generally involved, its cells being reduced in size and number and the nerve fibres more or less wanting. The lateral columns are sclerosed but the damage does not correspond accurately with the crossed pyramidal tract. The lesion diminishes from below upward and is chiefly in the external portions. Lissauer's zone is often affected. The bulb and pons are sometimes small and the bulb may contain degenerated nerve fibres. The brain is unaffected. Apparently the disease is a combination of degeneration and sclerosis, and the sclerosis is intermediate in character between posterior sclerosis and ataxic paraplegia or insular sclerosis. Sclerosis of the posterior columns, alone or with sclerosis of the pyramidal tracts, and cerebellar hypoplasia may be present as isolated lesions or more or less combined. Possibly the cases following infective disorders are toxic in origin, not dependent on abiotrophy, and not familial. The hereditary cases are perhaps due to arrested development of some nerve tracts in foetal life and slowly progressive degeneration.

*Diagnosis.*—This disease has to be differentiated from tabes dorsalis and ataxic paraplegia, both rare in childhood. The various types of cerebellar and spinal hereditary ataxia must be distinguished from each other and from cases of post-natal origin.

*Course.*—The earliest symptoms are disorders of gait, hyper-extension of the great toe, loss of knee jerk, and sometimes disturbance of speech. Ataxia develops in 3-5 years and is progressive. Finally the patient is unable to walk, has difficulty in using the fingers, is confined to bed and may be completely crippled, speech becoming unintelligible. Remissions and aggravations occur but it is steadily progressive. Recovery is unknown. The duration is indefinite. Death results from marasmus, intercurrent disease, or suddenly from bulbar palsy.



*Treatment.*—Maintain the general health and nutrition, making use of massage, passive movements, galvanism and exercise. Provide a support for the spine.

**Hereditary Spastic Spinal Paralysis.**—Spiller (1902) reported 14 cases in 5 generations of 1 family. It does not always begin in childhood. It is more common in males than females. The child becomes easily tired, awkward in movement and drags the feet. Later signs are spasticity, rigidity, pes equinus, and bending forward of the trunk; increased difficulty in walking without actual paresis; persistent spasm, even while at rest; exaggerated reflexes, ankleclonus, Babinski's signs and hyper-extension of the big toe; and generally some diminution of intelligence. The course is tedious and after death there is found sclerosis of the lateral tracts, and to a slight extent of the lateral cerebellar tracts and columns of Goll.

*Family Myopathic Sclerosis* is a sub-variety with progressive muscular atrophy and degeneration of the motor nerve cells in the cord and bulb.

**Tabes Dorsalis.**—Like general paralysis this is the result of congenital syphilis and not a family disease. In rare instances it is due to syphilis acquired in the early months of life. It differs from Friedreich's disease in the presence of a pre-ataxic stage, and the absence or late development of inco-ordination. It affects both sexes equally; and begins at 8-20, commonly at 10-12 years of age, in 1 instance (Marburg, 1908) at 2 years. The early signs are enuresis, lightning pains, amblyopia and gastric crises; and weakness, sluggish knee jerks, Argyll-Robertson pupils, sometimes loss of knee jerk and optic atrophy, paræsthesiæ of the legs and feet. Later symptoms are absent knee jerks, unequal pupils, Romberg's sign and slight ataxia. In the infantile type bladder and micturition troubles are the first indications and are followed by ocular and sensory symptoms, ataxia being absent. About 100 cases are on record. Some end in general paralysis. The progress is occasionally rapid. Usually it is slow and the disease remains stationary for years.

**Disseminated Sclerosis.**—*Syn.: Insular or Multiple Cerebral and Spinal Sclerosis.*—The existence of the adult type of this disease in children is doubtful. Recorded cases have generally followed an infective disease, notably measles, scarlatina and diphtheria. Probably these are cases of diffuse encephalitis or encephalomyelitis with secondary connective tissue proliferation. Others are perhaps of the nature of a diffuse sclerosis or focal myelitis from congenital syphilis. The chief symptoms are scanning speech, nystagmus, intentional tremor, disordered gait, exaggerated knee jerks and epigastric reflex, and mental deficiency; sometimes defective ocular movements, bladder weakness and sensory disturbances. True multiple sclerosis, with proliferation of the neuroglia, is possibly a diffuse gliosis and analogous to a neoplasm.

**Paralysis Agitans.**—Lannois (1894) reported a case in a boy, 11 years old. Tremor developed a few months after measles. From 12-18 years



of age he did not grow. The tremor increased, was exaggerated by emotion, heat, cold and voluntary movement, and absent during sleep. It affected the whole body, chiefly the upper limbs, but not the face. He had a "Parkinsonian" attitude on standing and walking. Possibly this was a sequel of mild encephalitis.

**Tumours** of the cord are rare, usually diffuse and inoperable. Some are tuberculous and end fatally from meningitis. Batten has reported diffuse sarcomatosis of the brain and cord in a boy, aged 10 years. During an illness of 9 months the symptoms were headache, vomiting, optic neuritis, loss of sight and hearing, exaggerated reflexes, leucocytosis, endothelial cells in the cerebrospinal fluid, convulsions, emaciation and flexor rigidity. The symptoms of tumour vary with the site.

**Peripheral Birth Palsy.**—*Syn.: Paralysis of the Newborn—Erb's Paralysis—Obstetrical Paralysis of Duchenne—Upper Arm Palsy.*—Erb's paralysis or birth palsy, to use the most common names, is a paralysis of one or both upper arms from injury to the brachial plexus during labour. It was ascribed by Danyau to forceps in 1851. Duchenne noted cases after difficult labour and Erb fully described it. Other varieties or modifications may occur from a like cause, e.g. paralysis of one or both lower arms, a lower arm type.

**Etiology.**—In my experience most cases have resulted from vertex presentations in primiparæ. Usually it is ascribed to extraction in breech presentation, the injury being inflicted by pulling on the shoulder to facilitate delivery of the head; pulling down the arm when it is above the head; elevation of the arm upward and backward, causing compression of the nerves between the head of the clavicle and the first rib or the transverse processes of the vertebræ, possibly aided by traction on the clavicle. In cranial presentations it may be due to the pressure of forceps on the lower nerves of the plexus; depression of the shoulder with the head bent to the opposite side and rotated; lack of flexion in face presentations; traction on the head producing strong pressure on the shoulder; or to traction on the neck by the finger or hook in the axilla. It has been noted after apparently normal labour, with no undue difficulty or instrumental interference. In a few instances it has been ascribed to constriction of the neck by the umbilical cord or in the pelvic passage during prolonged labour.

**Site of the Lesion.**—The damage is located in the fifth cervical nerve (Wilfred Harris); in the sixth cervical, and the fifth to a less extent; in the fifth and sixth always, seventh frequently, eighth and first dorsal occasionally (Clark, Prout and Taylor, 1905). In 5 operation cases Kennedy found the damage involved the junction of the fifth and sixth cervical nerves, anterior divisions; complete rupture in 4 and cicatricial pressure in the fifth. This may be accepted as the common site, namely,



at or just below the junction of these 2 nerves, below the origin of the nerves supplying the rhomboids and the spinati.

The *nature of the damage* is a complete rupture, rupture of a few nerve fibres, rupture or stretching of the nerve sheath and fibres with laceration and hæmorrhage, interstitial hæmorrhage, direct or indirect compression or bruising of the nerves. Cicatricial changes and neuritis are secondary. The axis cylinders are destroyed and new connective tissue forms between their ends, showing under the microscope strands of scar tissue among intact bundles of nerve fibres.

The *muscular affection* depends on the extent of the damage and on anatomical variations in plexus formation. In an uncomplicated case of fifth root palsy the deltoid, spinati, biceps, brachialis anticus, supinators and radial extensors of the wrist are affected. The pectorals, latissimus dorsi and triceps suffer if the sixth nerve is involved; and the extensors of the fingers if the seventh nerve suffers. In avulsion of the whole plexus the limb is entirely paralysed. The pectorals and latissimus are partly supplied by the eighth nerve. The "lower arm" type of palsy is usually the remains of total arm palsy but may occur independently. In 3 reported cases the cause was over-extension of the head in face or chin presentations. The seventh cervical, and to a less extent the eighth cervical and first dorsal nerves, were damaged far enough from the cord for the ocular fibres of the sympathetic in the first thoracic root to escape. A bilateral upper arm palsy under my care was associated with paralysis of the sterno-mastoids and diaphragm. The trapezius was also involved. As a rule the deltoid is the muscle chiefly affected, then the biceps, brachialis anticus, supinator longus, and less frequently the spinati and the coraco-brachialis. Occasionally the deltoid suffers alone.

*Symptoms.*—Severe cases attract immediate attention. Milder ones may not be noticed for some weeks. The arm hangs down loosely by the side of the trunk and is not moved. It is close to the trunk and rotated inward; the forearm extended, pronated and incapable of flexion or supination; and the hand in the "tipping" or "policeman" position, turned back and somewhat flexed. Sensation does not suffer, except in severe cases in which there may be anæsthesia over the outer part of the arm, over the area supplied by the circumflex and external cutaneous nerves. A slight degree of anæsthesia is unrecognisable in infants. Wasting of the muscles comes on in a few weeks. The muscles are so small and well covered with fat in babies that wasting is not noticeable for a considerable time. In bad cases the reaction of degeneration may be present. Rigidity may occur early from unopposed action of the opponent muscles, and later from contraction of the affected ones. Involvement of the sympathetic causes narrowing of the palpebral fissure, retraction of the eyeball and myosis. The injury may be complicated by fracture of the



humerus or clavicle, separation of the head of the humerus, facial palsy or wry-neck.

*Diagnosis* is easy in the upper arm type dating from birth. Cerebral palsy is rarely monoplegic and still more rarely limited to one group of muscles. Epiphysitis, separation of an epiphysis, fracture and dislocation of the clavicle must be excluded. In these affections there is pain, no true paralysis, and no electrical changes. At a later age it may be impossible to be certain whether the palsy is a "birth palsy" or due to poliomyelitis of the fifth segment of the cord. The history and grouping of the paralysis are the most reliable features. Similar palsy may result from wounds, pressure of enlarged glands or tumours, neuritis and spinal caries.

*Prognosis.*—Mild cases show little or no discomfort and tend to recovery. In more severe ones the child is peevish and irritable; and the signs of pain and anæsthesia indicate considerable damage and laceration of the nerve elements. The outlook is nevertheless fairly good in these severe cases, but they may last for years or be permanent. Partial recovery is common. Complete rupture, with separation of the divided ends and cicatrization, is almost certain to be incurable without operation. There is little prospect of recovery unless signs thereof are present by the ninth month. Mild cases begin to improve in 1-3 months and are well in a year. Flexion of the elbow is the first sign of improvement and may be the only one. The deltoid recovers last. In the presence of R.D. recovery will be slow or absent, yet it may be complete. If the muscles respond to faradism recovery is rapid. Some writers regard the prognosis as much more grave. Rotch states that most cases never recover and that partial recovery cannot be expected for years. Warrington and Jones (1906) say that 30-40 per cent. end in useful recovery. Atrophy, contractures and maldevelopment of the arm, shortening of the tendon of the subscapular muscle, and sometimes subluxation of the head of the humerus, are the chief sequels.

*Treatment.*—Keep the arm at rest, to allow time for repair of the damaged tissues. Wrap it in cotton wool and fix it to the side, as in fractured clavicle. Keep overstretched muscles mechanically relaxed for months, e.g. wrist drawn by a sling toward the neck to keep the flexors of the forearm relaxed, and hyperextension of the wrist if the extensors are paralysed. Maintain the nutrition of the affected muscles by cutaneous friction, massage, salt baths, exercises and electricity. Use the weakest available faradic current, if the muscles react, and make each muscle contract 20 times. Use galvanism, making and breaking the current, if there is no response to faradism, but replace it by faradism as soon as faradic irritability returns. At first apply galvanism daily, 5 milliampères for 5-10 minutes, with the arm and the negative pole in a water bath and the positive pole between the shoulders. After a



few days make the muscles contract daily and later on only on alternate days.

Operation has been successfully done. The damaged portions of the nerve are excised and the divided ends sutured. It must be advised as soon as it is evident that recovery will not take place. The younger the child, the better is the result. It must not be done before the third month of life and not then, if improvement is progressive. It is preferable to wait until 1 year of age for the risk of shock is less, the field for operation larger, the amount of paralysis defined, and the cicatrix localised. Marked benefit has accrued as late as 14 years of age (Kennedy). At the operation test the exposed nerves by electrical stimulation to ascertain their distribution. Nerve anastomosis, crossing and transplantation have all been adopted. Massage and electrical treatment must be continued afterward.

**Radial Palsy** or paralysis of the musculo-spiral nerve causes palsy of the extensors of the wrist, wrist-drop and flexion of the fingers. It may be congenital, due to an amniotic band or intra-partum pressure on the nerve; or acquired, due to lead poisoning, pressure during sleep, or fracture of the humerus. The *crural nerve* may be damaged at birth and cause a "lower limb" birth palsy. *Peroneal palsy* may be due to traumatism, or damage at birth. *Paraplegia* may arise from injury to the spine and cord in difficult labour; and possibly some of the bilateral birth palsies ascribed to injury of the sixth cervical nerves, notably those in which the sterno-mastoids are involved, really depend on injury to the spinal cord.

**Pressure Paralysis.**—*Syn.: Ischæmic Paralysis or Myositis — Volkmann's Contracture.*—If bandages or splints are applied too tightly, the blood supply to the subjacent muscles is interfered with and they become swollen by œdematous infiltration. The extremity of the limb swells and the whole limb is painful or uncomfortable. In the forearm, in which it is most frequent, the hands and fingers may be discoloured, blue and swollen. Often pressure sores are present on the skin, usually near the bend of the elbow. The muscles appear paralysed and the fingers and wrist become flexed, perhaps within a few days. The nature of the contracture depends on the flexors being stronger than the extensors. There is no true paralysis and no sensory changes. It is generally a sequel of fracture about the elbow joint and rapid in onset. The forearm is pronated and slightly flexed at the elbow. The wrist is markedly flexed and the metacarpo-phalangeal joints are extended. The phalangeal joints of the thumb and fingers are so flexed that the fingers are often buried in the palm. If the wrist is extended, it is impossible to extend the fingers. The muscles can be moved to an extent limited by the degree of contracture. Subsequently the muscles undergo interstitial fibrosis, becoming smaller, harder, and contracted. W. Harris



(1908) found, in 7 out of 9 cases, severe neuritis of the median and ulnar nerves with anæsthesia, wasting, and R.D. in the intrinsic muscles of the hands. The R.D. may be present in the flexors of the wrist and fingers. It is therefore a combination of myositis and neuritis in many instances. Treatment is of little value if fibrosis has taken place. Warm baths and massage twice a day may prove beneficial, and possibly the injection or inunction of thiosinamine or fibrolysin. Surgical measures are required for the relief of deformity.

**Facial Palsy.**—As a birth palsy facial paralysis results from the pressure of forceps, fracture of the base of the skull, or from pressure by a faulty pelvis or intra-pelvic tumour in prolonged labour. Subsequently it is due to meningitis, tumours, mischief in the ear, or causes affecting the nerve after its exit from the stylo-mastoid foramen. In rare instances it is associated with paralysis of the sixth or eighth nerve, possibly from nuclear degeneration.

*Bell's palsy* is the acquired variety, affecting the frontal and inferior branches of the nerve. It is due to cold, pressure, injury and other causes of neuritis, or middle ear disease. Cases due to cold often show a family tendency, several children becoming affected. They are rare in the first 2 years of life. So-called "rheumatic" cases are sometimes due to transient acute otitis media. Infants are less susceptible than older children to facial palsy from ear disease, for the inflammatory products cannot exert the same amount of pressure in an incomplete bony canal. Hensch and Goodhart regard this combination as pathognomonic of tuberculosis, but it may occur independently. Caries or necrosis of the aqueductus Fallopii is followed by extension of the destructive process to the nerve, or a simple inflammatory effusion in the aqueduct may compress the nerve. Facial palsy in chronic otorrhœa is a grave sign for it shows necrosis of the temporal bone is present.

If the cause is at the base of the brain, the auditory and other nerves may be involved and there are cerebral symptoms; if in the geniculate ganglion, there may be disturbance of lachrymation and palsy of the soft palate; if external to the ear, the signs are those of a pure motor palsy. Bilateral facial palsy is rarely due to ear disease, and is more likely to depend on congenital syphilis and be associated with or followed by deafness.

The *prognosis* depends on the cause. Pure motor palsy from injury at birth or cold is rarely permanent, though I have known it persist in a mild form in a case which came on during infancy. The outlook depends on the electrical reactions during the first 2 weeks, e.g. diminished irritability, R.D., or absence of response. Many aural cases recover completely, others incompletely. Recovery is rare, if the canal is necrosed; usual, if the nerve is only compressed by effusion. Conjunctival irritation, from imperfect closure of the lids, and herpetic eruptions are occasional sequels. The treatment consists of persistent galvanism with a current of 3-5



milliampères and facial massage. Nerve anastomosis may be tried in permanent cases.

Other *cranial nerves* are rarely affected except as the result of poli-encephalitis inferior or bulbar disease. A periodic *oculo-motor palsy* of one or more nerves, partial or total, may be associated with migraine and last for days or months (p. 764).

*Polyneuritis* is described under the head of diphtheritic palsy, and may follow other infections. Occasionally it is epidemic, a variety of acute anterior poliomyelitis, or due to lead, arsenic and alcohol.

**Sympathetic Nervous System.**—Paralysis or irritation of the sympathetic nerves in the neck results from the pressure of glands, thyroid, etc., injury at operation, disease of the spinal cord, first dorsal and eighth cervical roots, injury of the brachial plexus at birth, syringomyelia and tuberculous meningitis. Paralysis causes contraction of the pupil and palpebral aperture, sometimes retraction of the eyeball (*enophthalmos*), redness and anidrosis of the affected side of the face. Irritation causes dilatation of the pupil and palpebral aperture, sometimes *exophthalmos*, and hyperidrosis.

General hyperidrosis occurs in neurasthenia, hysteria, Graves' disease and epilepsy. It is localised in hemiplegia, affections of the spinal cord and tuberculous meningitis. It may be paradoxical, occurring under conditions which normally inhibit it; or abnormal in distribution and unilateral. It may be limited to the hands, feet, tip of the nose or forehead. For this variety ergot is sometimes useful. Chilblains, Raynaud's disease, angio-neurotic œdema, migraine, syncope and swooning, epilepsy and eclampsia may depend on the disorders of the sympathetic nervous system which induce vaso-constriction or vaso-dilatation.



## CHAPTER LV.

### THE MUSCULAR ATROPHIES.

*Werdnig-Hoffmann Type—Peroneal Atrophy—Pseudo-hypertrophic Paralysis—Facial Type—Erb's Juvenile Type—Amyotonia Congenita.*

The Myopathies as distinguished from myelopathies are characterised by the absence of definite changes in the spinal cord. In recent years there has been a tendency to favour the view of a spinal origin. There is no sharp line of demarcation for intermediate types occur. Degenerative changes in the cells of the cord in cases of long standing are probably secondary. These diseases are essentially congenital or endogenous in that they depend upon some defective tendency in development of the muscular tissues. The tendency is a potential one and the atrophy may not develop for many years. Excluding the spinal and neural types it is an inherited weakness in the muscle fibres. It affects more males than females and is transmitted by the mother. The same type occurs in members of the same family. Groups of muscles of the same period of development are affected simultaneously. The onset is during childhood, sometimes in adolescence. Perhaps there is a past history of awkwardness and difficulty in learning to walk.

The general features are slow progressive paralysis in muscles or groups of muscles, and atrophy or hypertrophy of those affected. The palsy is flaccid in type and fibrillary contraction is absent. There is no qualitative electrical change nor a decreased reaction. Muscular tone is often present and the tendon reflexes are preserved for some time. Foot contractures are common. There is no affection of muscles in groups corresponding to groups of cells in the cord. The extrinsic muscles of the eyes and the muscles of the larynx, pharynx and diaphragm are never attacked. The course is slow and the patients eventually become bedridden. Death is due to tuberculosis or intercurrent disease.

The essential change is a wasting of muscle fibres, which may or may not be associated with an overgrowth of interstitial tissue or a marked tendency to deposition of fat. The muscle fibres undergo granular degeneration, and the muscles are pale red or yellowish in colour. At first the muscle is not uniformly affected. The wasting depends upon the congenital defect rather than on pressure from overgrowth of the interstitial tissue, for such overgrowth may be entirely absent. The loss of power is in



proportion to the atrophy. Possibly in some instances there is a quantitative as well as a qualitative defect, and the muscle may be smaller than normal.

*Classification.*—The division into 2 groups of (1) Simple Atrophy, and (2) Atrophy with Hypertrophy, or Pseudo-hypertrophic Paralysis, is unsatisfactory. In some cases of pseudo-hypertrophic paralysis there is no increase in the bulk of the muscle and there are intermediate types showing connection between these varieties. The following classification shows the connection between the spinal, neural and purely muscular affections :—

A. Spinal Type or Progressive Muscular Atrophy.

(1) As seen in adults (rare).

(2) Early Infantile, or Werdnig-Hoffmann Type.

B. Peroneal Atrophy ; a neural type.

C. Muscular Dystrophies.

(1) Pseudo-hypertrophic Paralysis,

(a) with hypertrophy ;

(b) without hypertrophy ; idiopathic muscular atrophy.

(2) Idiopathic Atrophy ; with wasting of nearly all the muscles, occasionally a little pseudo-hypertrophy, and probably a variety of the previous group.

(3) Amyotonia Congenita ; an infantile or congenital type.

(4) Facio-scapulo-humeral Type of Landouzy-Déjérine (Facial Type).

(5) Erb's Juvenile Type, possibly a variety of the last group.

(6) Intermediate or combined forms.

Many of these types may occur in different members of the same family. The facial type and Erb's juvenile form are more frequently associated than the others. All types are connected by intermediate forms with pseudo-hypertrophic paralysis. The latter disease and the peroneal type breed most true. In the muscular dystrophies the extrinsic muscles of the hand are rarely affected. It is rare for any one variety to occur sporadically or to be limited to one generation. The sexual distribution is variable. Sometimes the females, more often the males, are most affected, perhaps because males do not live long enough to transmit the disease. It may be transmitted by unaffected females. In other instances both sexes suffer equally. Consanguinity may be a cause. Usually no direct exciting factor can be found ; a few cases have followed infective fevers.



**Werdnig-Hoffmann Type.**—This is an infantile progressive muscular atrophy of spinal origin. About 30 cases are on record. It begins at 6-12 months of age with weakness of the legs, back, neck and shoulder muscles, and then those of the hands and feet. Gradually complete paralysis ensues with atrophy, rarely pseudo-hypertrophy, disappearance of knee jerks and sometimes fibrillary twitching. Intelligence, sensation, speech, cranial nerves and the sphincters are unaffected. Death takes place in 1-4 years from involvement of the respiratory muscles, pneumonia, or enteritis. The anatomical changes are atrophy of the cells in the anterior cornua, degeneration of the root fibres and motor nerves, and muscular atrophy.

The *hereditary type of Leyden* is probably of the same nature. It attacks many members of the same family, is more common in males, and usually begins at 8-10 years of age. The early signs are weakness and wasting in the muscles of the back and lower extremities, lordosis and difficulty in walking.

**Peroneal Atrophy.**—*Syn.*: *Spinal Neuritic Atrophy* (Bernhard)—*Progressive Neural Muscular Atrophy* (Hoffmann).—This type was fully described by Tooth (1886) in a thesis entitled “The Peroneal Type of Muscular Atrophy”; and by Charcot and Marie in the same year. Many cases had been reported previously though the special characters had not been recognised.

The disease is hereditary, familial and transmitted indiscriminately by both sexes. Herringham traced it through 5 generations, 20 males being probably affected but no females. The disease was transmitted by healthy females to the male children. Affected males did not transmit to their children, and only to their grandchildren through healthy daughters. In this family the mode of inheritance was similar to that of pseudo-hypertrophic paralysis. In A. J. Whiting's group (1909) transmission took place from the affected to children of the opposite sex. The usual sex incidence is 1 female to 2 males (1-5, Sainton). Several members of a family are generally affected and isolated cases are rare. A sporadic case in a girl, 7 years old, was possibly a sequence of measles at 6 months of age. It commonly begins in the second half of childhood, usually before 10 years of age, and rarely after 20 years. Sainton gives the limits of age as 2-40 years.

*Symptoms.*—The earliest symptom is weakness and a tendency to “drag the leg” or “turn in” the feet. The atrophy is first conspicuous in the peroneal regions and then spreads to the other muscles of the upper and lower extremities. Sometimes it begins in the extensor longus pollicis, the extensor communis digitorum, the peronei, the intrinsic muscles of the foot, or even the gastrocnemius. Several of these muscles may be affected simultaneously. Later on the anterior tibial group of muscles and those of the calf and thigh, especially the vastus internus, are involved. After many years, and in rare instances at the same time, the hands are affected.



Here it begins in the muscles of the thenar and hypothenar eminences and the interossei. It then spreads to the muscles of the forearm, either extensors or flexors suffering first or most. The facial muscles, and usually the supinator longus and the muscles of the shoulder, neck and back escape. Ultimately the muscles of the pelvic and shoulder girdles may be involved. The disease is generally symmetrical. Club-foot or pes cavus results early from unequal involvement of the leg muscles. The *main-en griffe*, a "claw-like" deformity of the hands, is due to the intrinsic muscles being affected. It is an extremely rare condition in early life from any other cause. Scoliosis is sometimes present early, perhaps due to unilateral club-foot.

Muscle reflexes are diminished or absent in the affected parts. Both the knee jerks and Achilles tendon reflexes are generally lost. The knee jerk may be present, if the thigh muscles are not affected, although the atrophy below the knees is extensive. The cutaneous reflexes show variable alterations. Electric irritability to faradism and galvanism is diminished earlier and to a greater extent than in muscular dystrophy. Faradic irritability soon becomes extinct and the reaction of degeneration is usually present at an early stage. Even muscles, which are not atrophied, can only be stimulated with great difficulty. Patients are remarkably insensitive to faradism, possibly on account of some peculiarity in the skin (analgesia).

Fibrillary tremor is usually present. Cutaneous sensibility is sometimes impaired or lost, especially where atrophy is greatest and on the soles of the feet. Pain, hyperæsthesia, coldness and blueness of the limb, increased sweating and spasmodic muscular contractions have occasionally been noted. There is no sphincter trouble.

The disease may remain stationary for years or even permanently; usually it progresses slowly with periods of intermission. Cases vary somewhat in type. In those described by Charcot and Marie both upper and lower limbs were profoundly affected. In 5 members of the same family, aged 7-28 years, there was marked atrophy of the intrinsic muscles of the hands and feet, pes cavus, and weakness of the leg muscles in some cases (Lewis Jones, 1898). Electrical examination showed extreme insensibility to faradism, very great loss of muscular excitability to faradism and slight diminution to galvanism. In 1, a boy aged 7 years, the analgesia to faradism and loss of excitability were greatest in the hands, although the muscles were supposed to be unaffected.

*Pathology.*—It is not an affection of the anterior cornua for there is no diminution in the number of cells. Some of them show an advanced state of chromatolysis. Other degenerative changes have been found in the cord, similar to those due to section of peripheral nerves or anterior nerve roots, and are probably secondary. The chief lesion is sclerosis of the posterior columns. There is evidence that some cases are due to idiopathic muscular dystrophy, some secondary to neuritis, while others may depend on a combination of the two factors. Thus it may be due to a congenital tendency to



nerve or muscle degeneration, which may be excited by toxins setting up a neuritis or acting directly on nerve or muscle. In favour of neuritis is its tendency to follow acute specific fevers, notably measles; a chronic peripheral neuritis has been found in two cases. The presence of fibrillary contractions and of the reaction of degeneration also supports this theory. On the other hand the hereditary and familial cases appear to depend upon premature failure of nutrition in the peripheral nerve fibres.

**Pseudo-hypertrophic Paralysis.** — *Syn.:* *Pseudo-muscular Hypertrophy—Lipomatous Muscular Atrophy.*—This disease is transmitted by females though the females often escape, whereas the males affected rarely live to propagate the disease. Children of the same woman by different husbands may suffer. Many children in the same family may be affected, sometimes only the males. Males are much more often affected than females and more severely. Isolated cases occur. The type may vary in different members of the family. It begins during the period of development and in 75 per cent. before the tenth year. In rare instances it begins as late as 18 years of age. In a case under my notice no symptoms were present until 21 years of age. Usually the child has been walking and running about for some years before it is noticed. In 25 per cent. it shows itself when the child first attempts to walk and in such instances there is often backwardness in acquiring this function; in a few cases the child has never walked. Sometimes attention is not drawn to the disease until after puberty, although the increase in size and the weakness of the muscles have been present previously.

*Morbid Anatomy.*—The spinal cord and motor nerves are usually normal. The muscles are abnormally small and atrophic; they may be just like masses of adipose tissue and exhibit under the microscope only a few atrophied muscle fibres with little or no transverse striation. Occasionally no trace of muscle can be found. Other muscles may show a large amount of interstitial connective tissue and a variable quantity of adipose tissue between the atrophied muscle fibres. The greater the amount of connective tissue the greater is the wasting in the muscles. When much adipose tissue is deposited the wasting is not as a rule so profound. In portions of muscles removed during life some muscle fibres have been found larger than normal.

*Pathology.*—The first obvious change is an overgrowth of connective tissue in which fat may or may not be deposited. Muscular atrophy is mainly primary, partly secondary to the interstitial growth, while the increase in bulk is due to the deposition of fat. In late stages the fat is reabsorbed.

*Symptoms.*—It commences with weakness of the lower limbs. Attention may be drawn to the disease because of clumsiness in walking, inability to walk, difficulty in maintaining the erect position, difficulty in rising from the ground after falling or from a low chair, or in going up stairs. The



muscular enlargement may have been present for some time, and may have been regarded with pride as a sign of muscular strength. It develops gradually and is conspicuous in a few years. It may go on increasing for a time and then diminish as the muscular atrophy and absorption of fat continue. The muscles have no longer the elastic feel of healthy muscles but are hard, wooden and inelastic, even though small. Some muscles show marked wasting without antecedent hypertrophy and in certain cases which no doubt belong to this same group none of the muscles show any increase in bulk (*Idiopathic Muscular Atrophy*). The muscles most commonly hypertrophied are the gastrocnemius, gluteus, deltoid, triceps and infraspinatus. Those which are atrophied are usually the biceps, latissimus dorsi, serratus, lower part of the pectoralis major, flexors of the thigh, and extensors of the leg. The calf muscles are often affected and may reach a very large size. Although so bulky they are very weak and the child is not able to raise himself on the toes. The intrinsic muscles of the hand usually escape and those of the forearm are rarely affected. The masseters are sometimes enlarged, but usually all the face and neck muscles escape. The erectors of the spine are generally involved. Hypertrophy of the infraspinatus and atrophy of the lower part of the pectoralis major are the two most common features.

The effects of the disease show themselves in the position of the body on standing, in the gait, and in the mode of getting up from the supine position. In order to enlarge the base of support the legs and feet are kept wide apart. The head is erect, shoulders thrown back, and belly protuberant. To counteract the weakness of the flexors of the hips there is marked lordosis in the lumbar region. This disappears in a sitting posture, the back becoming convex backward from weakness of the erector spinæ muscles. The gait is peculiar and waddling. The feet are kept wide apart and the body sways from side to side so as to bring the centre of gravity over each foot in turn, while the pelvis is tilted to enable the child to swing the foot clear of the ground. The child is liable to fall if any obstacle is met with. In order to rise from the supine position the child first turns on the side or face. He then raises himself on his hands and feet, lowers the head, and gradually pushes the body and hands backward until he is able to get first one and then the other hand on the thighs. Having done this he is able to push himself up to the erect posture by climbing with his hands up the thighs. Or instead of getting on to the hands and feet he gets first on to the hands and knees, next places his hands on the thighs, then plants his feet on the ground and proceeds to climb.

Knee jerks may remain normal if there is no disease of the extensors of the knee, especially the vastus internus. If these muscles are affected the knee jerk is gradually abolished. The electrical reaction is normal at first but as the wasting progresses the response to galvanism and faradism diminishes. There is never a reaction of degeneration. There is no



fibrillary muscular contraction, no disorder of sensation, and no affection of the sphincters. Mentally the child may be ahead of his equals in age, but in some cases there is an associated mental weakness or imbecility, from defective development of the brain.

Deformities arise from contraction of the affected muscles, or from unopposed action of the muscles which are little or not at all involved. The knees and elbows may become contracted in the flexed position. Talipes equinus results from contraction of the calf muscles and much deformity of the ankle joint may ensue. Lateral curvature is not uncommon. Epilepsy and vesical complications have been recorded.

The state of the muscles varies in different cases. They may be large or small; or one, or a few, may be large and the remainder small. In other cases the muscles may be small and atrophied from the onset. Gradually the affected muscles waste and the weakness increases until the patient is unable to walk or stand, becomes bedridden, and dies from some intercurrent disease, usually bronchitis, broncho-pneumonia, pneumonia or tuberculosis. Lung affections are liable to develop from the enfeeblement of the respiratory muscles. The progress is slow and irregular. Periods of intermission are not uncommon and at times there may be definite improvement. After puberty it progresses more slowly, but when once the patient is confined to bed it rapidly increases and deformities become more marked, until he is quite helpless except for some power in the hands. It is less severe and more slowly progressive in females. Sometimes it advances rapidly and rarely remains quite stationary. If it begins before puberty the patient is unlikely to attain adult age.

The *diagnosis* is based on the muscular weakness and enlargement, the age, the peculiar gait and method of getting up from the ground, and the absence of fibrillary contraction and the reaction of degeneration. The hands and forearms rarely suffer. Muscular weakness is of more importance than change in volume, especially if it has come on gradually or has started in infancy. Enlargement of the calf muscles, with contracture which cannot be overcome, is very significant, and importance must be attached to the enlargement of the infraspinatus with weakness of the pectorales and latissimus dorsi. It must not be forgotten that muscles may be badly affected without there being any actual alteration in size, but they feel very different from healthy ones. The peculiar method of rising from the ground is not pathognomonic and may occur in other affections in which there is weakness of the extensors of the hips and knee. Care must be taken not to confuse the disease with spastic paraplegia or infantile paralysis.

*Treatment.*—In order to maintain and develop the nutrition of the unaffected muscles fibres the patient should continue muscular exercise, short of fatigue, as long as possible. Carefully regulated gymnastic exercises are useful. Massage may help nutrition. Electrical treatment is of little or no value, and no advantage is obtained from drugs beyond that derived



from tonics such a cod-liver oil, iron and arsenic. Tenotomy may be necessary to remedy deformities which interfere with walking. The general health must be maintained, and the patient guarded against exposure to cold and other causes of lung affections and intercurrent disease.

**Facial Type**, or *Facio-scapulo-humeral type* of Landouzy-Déjérine, called by Duchenne "*Progressive Muscular Atrophy of Infants*."—This has been traced through 5 generations. It seldom begins in childhood but may start as early as 2 years of age. Usually it commences in the second decade, or not until the fourth decade of life. It begins in the orbicularis muscles of the eyes and mouth and the masseters, and extends to those of the arms and shoulders. It is usually symmetrical and the face acquires a characteristic dull, expressionless look, the "myopathic face." The atrophy of the orbicularis oris causes separation of the lips, inability to pout and imperfect labial articulation. Sometimes the lip muscles are hypertrophied. The smile is altered from paresis of the levator labii superioris and the zygomatics. The buccinators, frontales and orbiculares palpebrarum are less often affected. The gluteal and thigh muscles may be wasted; those of the forearm and leg are well developed. The sphincters are unaffected and the reflexes are lost if the atrophy is extreme. It may be limited to the muscles first attacked or spread slowly, with intervals of years between each extension. Death is due to intercurrent disease.

**Juvenile Type of Erb**.—This differs from the facial type, for the face is unaffected and occasionally hypertrophy occurs. It begins in late childhood or early youth with progressive wasting and weakness of certain groups of muscles. Usually it begins symmetrically or unilaterally in the muscles of the upper arm and shoulder, namely those affected in pseudo-hypertrophy. Thus it may begin in the lower part of the pectoralis major and the latissimus dorsi. It involves the biceps, triceps, supinator longus, upper part of the pectoralis major, pectoralis minor, trapezius, and rhomboids; less frequently the serratus magnus and spinati, rarely the deltoid. The other muscles of the forearms and hand and those of the leg may remain intact for a long time. The muscles of the pelvic girdle, thigh and back are usually affected, especially the extensors of the hip, flexors of the knee, and less often the glutei. In some cases the lower limbs suffer but little, and in others the peroneal type of atrophy is present. In late stages the intercostals and diaphragm may suffer, and rarely the abdominal muscles. It may be associated with pseudo-hypertrophy of some muscles, especially the deltoid and infraspinatus.

**General Diagnosis**.—These muscular dystrophies are all apparently of the same endogenous causation and differ merely in their distribution, age of onset, sex liability and mode of transmission, and the relative distribution of atrophy and pseudo-hypertrophy. In all cases the electrical reactions are unaltered or merely quantitatively diminished. There is no reaction of degeneration. There is no fibrillary twitching. There is no



sensory disturbance or sphincter trouble ; the intrinsic muscles of the hand and muscles of the forearm escape or remain intact for a very long time ; and the knee jerks are unaffected, or lost if the thigh muscles are atrophied. They show distinct differences from progressive muscular atrophy of spinal origin and from the neural form as seen in the peroneal type. In every case the course and prognosis are the same. The treatment is that advised for pseudo-hypertrophic paralysis. Intermediate varieties occur in which pseudo-hypertrophy or muscular atrophy is combined with trophic changes in the anterior cornua.

**Amyotonia Congenita.**—*Syn. : Myatonia, Hypotonia or Atonia Congenita—Congenital Muscular Atrophy—Congenital Amyoplasia.*—This affection was described by Oppenheim in 1900. It differs from myopathy in several features. There is no hereditary tendency, no familial distribution, and no true paralysis. Silvestri (1909) reported a case at 45 days of age. A maternal aunt died of progressive muscular atrophy. The mother developed osteomalacia in the eighth month of pregnancy. Her first child showed a similar condition at birth, recovered partially and at 16 years presented Erb's juvenile paralysis. The fourth child was myxœdematous. The affection is present at birth. It has developed in the first year of life after an acute illness and after bronchitis at 1 year of age. It may be general, chiefly or entirely in the legs, or limited to the head and trunk. There is no tendency to spreading.

The muscles, especially those of the legs, are thin, flabby and weak, soft and atonic, but not truly atrophic. The weakness varies with the size. The neck, trunk and intercostal muscles may be affected. Occasionally the arm and trunk muscles escape. The periphery of the limbs is most affected, including the intrinsic muscles of the hand if the arm is involved. The diaphragm, sphincters, and muscles supplied by the cranial nerves are not involved. Inability to close the eyes has been noted in 3 out of 28 cases on record (1907-8). There are no sensory changes, no trophic changes, no fibrillary tremor, no reaction of degeneration, and no disorder of the sensory organs or brain. The electrical reactions and the reflexes are weak or absent.

The child is unable to stand or walk, and assumes a squat, frog-like or huddled up attitude. The limbs are flaccid and motionless. The loss of power varies from impaired voluntary action up to almost total functional paralysis. Weak voluntary contractions can be detected. The joints are flail-like from hypotonia and flaccidity, so much so that it may be possible to hyperextend the legs on the abdomen until the heels lie behind the head, or to hyperextend the foot until the dorsum touches the shin. Other joints are similarly mobile. Contractures have been reported in the lower limbs. The mental state is normal.

**Morbid Anatomy.**—In a child aged 22 months no changes were found by Spiller (1905) in the nervous system. The muscles were small and pale.



Under the microscope they had a hyaloid appearance with much fatty connective tissue, small muscle fibres and indistinct longitudinal striation. Baudouin (1907) found, in a child of 4 months old, deficient myelination of the anterior lumbar roots and various nerves. The muscles were very pale, embedded in fat, and contained an excess of connective tissue, the muscle fibres varying in size. Collier and Holmes (1909) found similar muscular changes, small anterior spinal roots and a marked deficiency and atrophy of the anterior horns. All these changes have been found in true myopathy. Possibly, the state of the nerve elements is one of delayed development.

*Pathology.*—The condition is ascribed by Oppenheimer to retarded muscular development and functional derangement of the anterior cornua; by Bing to arrested development of the medullo-cerebellar tracts; and by Baudouin to a defect in the peripheral neuron. It has also been ascribed to defect in the thymus, and regarded as akin to myxœdema.

*Course.*—It appears to be non-progressive and improvement may be hoped for. The upper extremities recover before the lower ones, and the less affected flexor muscles before the extensors. The knee jerks may return after prolonged absence. There is grave liability to fatal bronchitis if the thoracic muscles are involved.

It must be diagnosed from rickets, infantile palsy, and myopathies. A case described by Guthrie under the name of “rachitis pseudo-paralytica hypotonia muscularis” was probably of this type. It may exist in a mild degree as “double jointedness.” The treatment consists in massage, faradism, baths, and the administration of arsenic and strychnia. Faradism produces the most rapid improvement.



## CHAPTER LVI.

### SPASMODIC FUNCTIONAL NEUROSES.

*Convulsions — Epilepsy — Tetany — Laryngospasm — Myoclonus — Habit Spasm — Tics — Nodding Spasm — Head Banging — Nystagmus.*

A condition of galvanic hyper-excitability of the peripheral nerves, associated with a tendency to convulsions and muscular twitchings, occurs in children, especially infants. It has been named the *tetanoid* condition, **Spasmophilia** or the *spasmophilic diathesis*. It was first found in tetany, and may be present in laryngospasm and eclampsia. The child is usually rachitic. This diathesis is more or less hereditary. Apart from the above-mentioned diseases, it is most frequent from November to April and at its maximum from February to April. The common age is 6-18 months. Fits and laryngospasm are most frequent from the fourth to the twenty-fourth month and tetany from the fourth to the thirty-sixth month. All these affections may occur at a later age.

It has been ascribed to hæmorrhage into the parathyroids (von Pirquet) and to some unknown metabolic disturbance dependent on the food supply, usually cow's milk. Whey is said to be equally injurious. It is rare in the breast-fed and often ceases on a diet of breast-milk. According to Gregor it can be cured by starvation and a liberal water supply. It occurs in both fat babies and in lean ones with alimentary troubles. Probably it is due to a toxin of intestinal origin. The characteristic electrical reactions are that K O C is below 5 ma. and that  $K O C > A C C$ .

**Convulsions.**—*Syn. : Eclampsia.*—A convulsion is a “motor discharge” which gives rise to an involuntary purposeless muscular spasm of temporary duration, as opposed to the constant muscular spasm resulting in permanent contracture. In generalised convulsions the spasms are tonic and clonic, and are usually accompanied by other symptoms:—namely, a cry, more or less profound loss of consciousness, upward and convergent squint, loss of control over the sphincters, and drowsiness, stupor or coma. Gowers has pointed out that convulsions are far nearer normal action than their startling character suggests. Nerve-structures hold a vast amount of latent energy in perfect readiness for instant relief, as is well seen in the sudden movements of a startled animal. The pre-disposing cause is an individual instability or an exaggeration of the normal instability of the nervous system in early life.



*Pathology.*—The actual mode of production is an involuntary discharge of nerve impulses from the nerve cells in motor areas of the brain. Such a discharge is caused by direct irritation of the motor cells, by stimuli transmitted from other parts of the brain, or by reflex irritation from other parts of the body. Sudden normal stimuli, e.g. light, sound, smell or touch, may cause a convulsion if the central nervous system is enfeebled or imperfectly developed. At birth some structures are perfect while others have to undergo considerable developmental change. Nerve structures lower in function, such as the reflex centres and motor tracts, are developed before the higher ones. Hughlings Jackson regards the central nervous system as containing sensori-motor cells grouped in 3 tiers, all parts of the body being represented in each tier. In infancy the upper and middle tiers are not fully developed, and the lowest one is in a state of unstable equilibrium and liable to respond unduly to exaggerated physiological stimuli. According to this theory *petit mal* depends on irritation of the frontal and occipital regions of the cortex; Jacksonian epilepsy, on a lesion in the Rolandic area or middle tier; and laryngospasm, spasmodic asthma, vomiting and possibly rigors, on irritation in the pons, bulb and cord.

The early predominance of the lower centres is slowly reduced into subjection as the higher controlling centres develop. The reflex activities of these lower centres are therefore unduly prominent in infancy, are under inefficient control, and react with abnormal violence to slight stimuli.

Defects, such as occur in imbeciles and allied cases, and delay of development from interference with nutrition, affect the higher undeveloped centres to a much greater extent than those structurally and functionally perfect.

All disorders of nutrition, e.g. rickets, and all causes leading to malnutrition result in the persistence of, or a return to, the infantile state with its imperfect control over the lower nerve-centres. The whole nervous system is affected, and convulsions readily occur and are almost invariably generalised. Localised or unilateral convulsions are commonly due to local cerebral causes. If due to an apparently generalised cause, the unilateral character can be explained by an unequal instability of the different motor cells, or an unequal distribution of the exciting factor by reason of circulatory or lymphatic inequalities.

Experimental results are not of much pathological value. Convulsions have been produced in rabbits by suddenly cutting off the blood supply to the brain, by mechanical irritation of the floor of the fourth ventricle, and by lesions of the sciatic nerve but not of the brachial plexus in pigeons and guinea pigs. Fits have been ascribed to vascular anæmia, such as follows severe hæmorrhage; and to vascular engorgement of the brain and its membranes, a condition frequently found after death, and possibly a result, not the cause. Engorgement of the right side of the heart and the lungs may be present as a result of the asphyxia rather than



the cause of the fit. Convulsions in the course of whooping cough are due to vascular engorgement, thrombosis, or hæmorrhage. There is rarely any evidence of causation after death except in cases of gross organic disease.

*Classification.*—On pathological grounds convulsions are divisible into groups according to their dependence on defective inhibition and reflex irritability, auto-intoxication, epilepsy, rickets, and spasmophilia. *Eclampsia infantum*, as a spasmophilia, must present the signs thereof between the attacks, no fever, and no evidence of disease or toxæmia. On anatomical grounds they are classified into:—(1) those in idiotic or imbecile children; (2) those in children with hemiplegia or secondary contractures; and (3) cases in which there is no organic nerve lesion, including eclampsia or *external fits*, and spasm of the glottis or *internal fits*.

*Inward convulsions* is a term often used for attacks of temporary pallor and apparently momentary unconsciousness. The child is still and rather stiff, moans and grins, with upturned eyes, clenched fists and thumbs flexed in the palms. The legs may be drawn up at frequent intervals. These may be mild attacks of laryngospasm and are sometimes called “holding breath spasms.” Occasionally they are slight syncopal attacks, due to colic or pressure of a dilated stomach on the heart, and less often truly epileptic.

*Agonal convulsions* are convulsive movements occurring just before or at the time of death and simply a mode of dying. It is due to the frequency of convulsive movements of this type that fits are regarded as common in infancy. *Screaming convulsions* are violent screaming attacks in infancy due to pain, usually colic, occasionally organic brain disease. Sometimes they indicate mental deficiency.

*Etiology.*—Idiopathic fits are those for which there is no apparent cause, and possibly epileptic. Symptomatic fits are due to cerebral causes, reflex, toxic, asphyxial, or circulatory in origin, or are a mode of dying. The more carefully fits are investigated in infancy, the rarer is the diagnosis made of an idiopathic origin. Toxic causes, e.g. fevers, uræmia, food toxins, poisons, alcohol and narcotics, are often difficult to recognise.

The predisposing factors, those which produce instability or irritability of the nervous system, are inherited or acquired. Slight causes will produce fits in the predisposed. The predisposing factors dependent on age and development are common to all infants, yet only a small proportion have fits. If the exciting cause can be discovered, the results of treatment are more likely to be satisfactory.

*Heredity.*—A family history of organic or functional nervous disease, a neurotic temperament, insanity, suicide, immorality or excessive drinking, renders an infant more liable to fits. In some families all or most of the children suffer from infantile convulsions. Undue prominence has been given to heredity on account of its influence on the production of epilepsy.



In a great many instances the children of such parentage do not have fits in infancy. Even when they do, it is possible that the underlying cause may be found in the mode of feeding rather than in the family history. Heredity has extremely little influence *per se* during the first 3 years of life. I could only obtain a family history of epilepsy, suicide or insanity in 5 out of 200 successive cases. It is possible that alcoholism in the mother may affect the child through the foetal circulation. That an alcoholic father can exert much influence on the germ plasm is most unlikely. If one or both parents are alcoholic, other factors are present which impair the nutrition of the child. There may be gastric disturbance in the mother leading to deleterious changes in the milk, insufficient food and clothing, and chronic or occasional neglect of the child. These factors are quite enough to account for the fits, without ascribing them to the hereditary influence of alcohol. Tuberculosis and other diseases of malnutrition in the parents exert a predisposing influence in that the child may be delicate at birth. Provided the parents are healthy little attention need be paid to consanguinity or great difference in their respective ages.

*Age.*—Most fits occur before the completion of the first dentition. Out of 300 successive cases 44 began in the first 6 months, 52 in the second 6 months, 76 in the second year, and 44 in the third year. No less than 216 were under 3 years, 27 in their fourth year, and the remainder under 12 years. From the fifth to the twelfth year there is little difference in the yearly age-incidence. In those who have already suffered, there is a slightly increased liability at the onset of the second dentition.

Undue importance has been attributed to *rickets*. Neither its bone changes nor craniotabes explain the convulsive phenomena. These are really due to the dietetic and other conditions which give rise to the rickets. The frequent coincidence of rickets and fits is because rickets develops at the age when the child is likely to have unsuitable food. Fits are not much more common in the second than in the first 6 months of life, and rather more frequent during the first than the second year, when rickets is most marked. It must be regarded as a predisposing cause, mainly on account of its complications and partly because it impairs the nutrition of the nervous tissues.

The *cerebral causes* are organic diseases of the central nervous system such as concussion, meningitis, encephalitis and sclerosis; hæmorrhage, thrombosis and embolism; porencephalus, microcephalus and hydrocephalus; tumour, abscess and parasitic growths. The affections leading to cerebral hemiplegia are the most common. Fright is a definite exciting cause in a few children.

*Asphyxia* explains some cases in which there is interference with respiration. The venous blood directly irritates the motor cells, e.g. cyanosis in the newborn and cerebral congestion during whooping cough.



Possibly some of the fits in whooping cough and hydrocephalus are due to changes in cerebral pressure.

*Fever* may be ushered in by, or associated with fits, though they are rare at the onset of specific fevers, unless the child has had them before. They may occur in any specific febrile infection, general infective disorders, throat affections and even herpes; through the action of the toxins or micro-organisms, rather than the height of the fever. Occasionally they are due to hyperpyrexia or heat-stroke. *Toxæmic conditions* include fevers, uræmia, burns, blisters, and food poisoning. Uræmic fits have been recorded in the first month of life. The importance of food poisoning cannot be overrated. In many instances the cause is not a mechanical one but lies in the absorption of intermediate products of protein digestion, or some toxin, produced in the food before or after ingestion, acting directly on the central nervous system. Poisons such as lead, alcohol and narcotics are occasionally to blame. In country districts poisonous berries and fungi are not infrequent causes.

*Gastro-intestinal disturbance*, inducing colic, is very common in infancy. The younger the child the more probably is some indigestible article of food the cause of the convulsion. Under 6 months of age the attack is frequently due to cow's milk, boiled bread and starchy food, especially proprietary foods consisting mainly of starch. In older children I have seen convulsions due to pork, bacon, haddock, bread and cheese, peas, currants, cherries, strawberries and other indigestible foods. The influence of diet decreases as age advances. Thus, the attack was ascribed to dietetic causes alone in 22 out of 31 infants under 6 months of age, in 10 out of 30 in the second half of the first year, and in only 8 out of 32 in the second year of life. A typical and severe instance was that of an infant brought up from birth on cow's milk and Mellin's food. Fits began on the seventh day. On a diet of humanised milk they ceased for a week, and then recommenced at the rate of 20-30 a day. Bromides and belladonna proved useless. At 9 weeks of age a modified milk mixture was ordered, containing only 0·5 per cent. of protein. The fits immediately ceased and did not recur.

Other sources of reflex irritation are dentition (p. 225), worms (p. 314), foreign bodies in various orifices, chiefly the ear and nose; boils in the external auditory meatus and diseases of the middle ear; inflammatory affections of the nose and throat, polypi and adenoids; a tight phimosis, irritation from retained smegma and balanitis; severe injury to cranial or spinal nerves, sudden and severe pain in any part of the body, burns, renal or intestinal colic, intestinal strangulation and retention of urine. Errors of refraction and astigmatism, over-pressure at school, examinations, anxiety, fright, shock, masturbation and menstrual disorders have all been blamed. Some of the causes mentioned may be partly reflex and partly toxic in their action. There is little doubt that too much importance is attached to reflex causes. That a reflex source of irritation is present, is no proof that



it is the exciting factor. I have never known phimosis, for instance, induce a fit, although it is invariably mentioned in text books as a cause. Fits have coincided regularly with teething and have recurred at the onset of the second dentition. None of the various factors is in my experience of much importance as an exciting cause in comparison with those dependent on diet. Mere overloading of the stomach is a trivial factor. The fit usually comes on when the food has passed into the intestine. Occasionally it is due to mechanical irritation of the pylorus by indigestible food.

*Symptoms.*—Premonitory signs are generally absent. A certain amount of importance can be attached to restlessness, anxious facies, twitching of the muscles of the face or eyelids, screwing up the mouth, and screaming attacks. All these may be due to emotion, flatulence, colic or teething. Signs of nervous irritability and gastric or intestinal disturbance must be regarded as warnings.

The onset is generally sudden. In a mild attack the face turns pale, the eyes are fixed and up-turned, breathing shallow, and a certain amount of rigidity supervenes. Then the child takes a deep breath and comes round. In severe attacks there are sudden pallor, unconsciousness, tonic spasms, clonic spasms, irregular breathing, cyanosis and sweating, followed by subsidence of the spasms, relaxation and return to consciousness. The muscular twitchings begin in the facial or ocular muscles, or in the limbs, and rapidly involve the whole body. The facial spasm causes violent contortion of the features, hideous grimaces. The head is retracted, the back arched; the elbows flexed, wrists bent, hands clenched and thumbs in-turned; the legs rigidly extended. General tonic spasms of the limbs ensue. A slight froth collects on the lips and clammy sweat on the forehead. The child is unconscious, with wide-open eyes and immobile pupils. Breathing is shallow, feeble and often spasmodic. The pulse is weak, frequent and perhaps irregular. Soon the face reddens, the lips become bluish; mucus collects in the throat and produces "the rattles"; and the bladder and rectum may be emptied, though the actual secretion of urine is suspended or scanty. Gradually the tonic spasms are replaced by clonic ones, which soon cease, and the child falls asleep. The cyanosis is due to inefficient breathing and obstruction to the free entry of air into the lungs by falling back of the base of the tongue.

The actual duration varies from a few seconds to several hours. A mild attack is soon over and the child seems normal in a few minutes. In the prolonged cases, the *status eclampticus*, the spasms may persist throughout but more often there are periods of relaxation and exacerbation. After an attack there is considerable prostration, muscular relaxation, and sometimes temporary paralysis of one or more limbs. Death is unusual, unless there is organic brain disease or a profound toxæmic exciting cause. Even a mild fit may prove fatal in a very young infant.



Sequels are rare, except temporary loss of power, aphasia, and mental dulness. Cases of amaurosis have followed frequent fits, with stupor or coma for some hours or days. This is sometimes called "*Post-eclamptic Blindness*," and has been ascribed to exhaustion or anæsthesia of the visual cortical centre, due to the fits. Out of 11 collected cases under 3 years, 8 were under 18 months, an age when basal meningitis is most common. In some of them the persistence of hemiplegia was suggestive of organic cerebral disease. The sight is usually regained.

*Diagnosis.*—There is no difficulty in recognising fits that are actually occurring but a diagnosis based on the history of the case is unreliable in slight attacks. A mild fit may be mistaken for syncope, and attacks of laryngospasm, faintness or colic ascribed to fits. Even the movements of thigh-friction are sometimes called fits. Age has an important bearing on the diagnosis of the cause. Eclampsia neonatorum is due to injury at birth, cyanosis or sepsis. Fits beginning after the second week of life are rarely due to injury at birth. In the pre-dentition period the common causes are diet, meningitis and otitis. Diet exerts a potent influence during the first dentition and rickets is often present. Subsequently, up to the second dentition colic and toxæmia are the most important causes. Alimentary disturbance or the ingestion of unsuitable food may be ascertained by inquiry and examination. Always ask what food the child has had during the previous 24 hours and inspect the vomit and stools, if any. Fever is a sign of acute disease or severe food poisoning. The cause of the fever may not be certain for a few days. In some cases there is obviously asphyxia or a disease likely to cause asphyxia. In doubtful cases look for albuminuria. A unilateral distribution occurs in Jacksonian epilepsy; prolonged unconsciousness, if there is an organic cerebral cause; and petit mal in serious cerebral defect. Epilepsy cannot be diagnosed with certainty during infancy. Stuporous sleep after a mild fit is suggestive. The presence of a definite morbid cause is in favour of eclampsia but, though the fits at first are eclamptic, they may pass into true epilepsy without any clear dividing line. Occasionally multiple fits occur for which no cause can be ascertained. Eclamptic fits due to spasmophilia may occur up to the eighth year of age and can only be distinguished from epilepsy by the evidence of spasmophilia between the attacks.

*Prognosis.*—The danger of a single convulsion or multiple attack is much overrated. Only in the very young, the very rachitic or the profoundly marasmic is death probable as the immediate result. Recovery ensued in the case of a child who was collapsed, sweating freely, with dilated pupils and no corneal reflex, pulse 156, respirations 72, T. 103-105° F. The convulsions had been going on for 12 hours and were due to cerebro-spinal meningitis. The prognosis depends on the cause, the duration, the age and the family history. Fits due to cerebral disease are the least amenable to treatment, and the prognosis is that of the primary affection.



In other cases the older the child the less likely is the attack to prove fatal. In late stages of fevers the outlook is serious for the nervous system is affected by toxins. Uræmic fits are not necessarily fatal although the child may be only kept alive by artificial respiration for several hours. If the fit is of gastro-intestinal origin, the child may never have another, provided it is properly dieted. During an attack the prognosis increases in gravity with the duration, the degree of asphyxia and general prostration, the feebleness of the pulse, and the depth of the coma. The outlook of status eclampticus is bad. The longer the interval between successive fits the better is the prospect, for drugs and other measures have a chance to exert their effects.

Excluding organic causes the prognosis is good as regards recurrence but varies directly as the hereditary predisposition. The probability of epilepsy is least when there is a definite cause. Infantile fits may be limited to a single attack; may be multiple and cease in a few months; may be continued into life-long epilepsy; or may cease and recur after puberty or later in life. The primary fit and the residual state of the nervous tissues, with its tendency to recurrent explosive discharges, are greatly influenced by heredity. Usually the tendency to fits dies out as the higher nerve centres acquire control over the lower ones.

Except in cases where the fits are very frequent, or very severe, and persist for a number of years it is uncommon for them to produce any effect on intelligence. After multiple fits a baby may remain drowsy and stupid for weeks and yet recover. Moral and general character are more likely to be affected than intelligence. The child becomes mischievous, destructive, irritable, bad-tempered, and perhaps a moral lunatic. The psychical defects may be due to maldevelopment or changes in the brain, which are the cause of the fits. Local convulsions are more likely than general ones to be due to organic brain disease. If no cause can be found and the fits are frequent, the prognosis is bad as regards life and subsequent mental development. It is advisable to be reticent in foretelling evil, in laying stress on bad family history and in diagnosing amentia. Enlarge on the favourable features of the case and the benefits of care and attention.

**Epilepsy.**—The epileptic fit is a type of convulsions of unknown pathology. According to Gowers 10 per cent. of the cases begin in infancy. The slight fits of infancy may pass into petit mal without the change being recognised. A diagnosis of epilepsy was made in 55 out of 300 cases of fits in children under 12 years old:—15 of them had fits during the first 2 years of life; 2 of these were more or less imbecile, and in 6 others there was a family history of mental affections. In only 13 out of the 55 was there a history of family predisposition. The earliest age, at which epilepsy was diagnosed, was 6 months and the subsequent course proved the diagnosis correct. In 44 out of 84 cases between the ages of 3 and 12 years the fits were regarded as epileptic.



The *etiology* is similar to that of infantile convulsions and includes all cerebral causes, congenital and acquired. A meningeal hæmorrhage at birth may be slight, produce little or no apparent damage, and only fits in childhood or later life. Too much importance is attached to infantile convulsions. If eclampsia is a spasmophilia and epilepsy is not, there is small probability of the transference of one into the other. Statistics are of little value. Fletcher Beach states that hereditary taint is present in more than one-third of the cases in the form of epilepsy, insanity, eccentricity or chronic neuralgia. Spratling found a family history of epilepsy, alcoholism, tuberculosis or insanity in over 50 per cent. of 1100 cases. True hereditary epilepsy is more frequently transmitted through the mother than the father, and may skip a generation. The chief exciting causes are alimentary disturbance and toxæmia. The foundation of the attack may be laid in school life or earlier, by unsuitable meals, constipation, or an excessive nitrogenous diet which unduly stimulates the nervous system.

*Idiopathic Epilepsy* assumes various forms. The name is given to those cases in which there is no apparent cause and no definite organic change. It begins at 6-15 years of age. Cases starting in earlier life are likely to be symptomatic or secondary. The patients are usually dull, stupid and unemotional, but in good health. The symptoms are much the same as in adults. A fit may be preceded for hours or days by irritability, excitability, depression, headache, giddiness or digestive disturbance. A definite *aura* is ascertained with difficulty in young children and is absent if consciousness is quickly lost. It may be motor, sensory, or psychical in character. A motor spasm or twitching usually starts in the thumb and spreads upward. Tingling or numbness may have a similar distribution. Epigastric discomfort, nausea or vomiting is the most common visceral aura. Vertigo, fear, hallucinations and illusions occasionally occur, and sometimes auditory, visual, olfactory or gustatory auræ. A girl, 9 years old, stated that an ugly bearded black man seized her by the right upper arm and cut her head off. A 14-year old boy felt thirsty before the fits. In *nocturnal epilepsy* the fits only occur during sleep, and the diagnosis depends on involuntary micturition or defæcation and drowsiness in the morning. Perhaps the tongue is bitten and small ecchymoses may be found in the skin.

*Haut Mal* is characterised by loss of consciousness, tonic spasms up to forty seconds duration, clonic spasms with lateral deviation of the head and eyes for perhaps 2 minutes, and complete stupor. The child may utter a sudden cry at the onset. Either the tonic or clonic spasm may be absent. The child recovers in about a quarter of an hour, or may not fully regain consciousness for about an hour and is then very irritable. Temporary paralysis and aphasia, automatism, mania, hallucinations and illusions are occasional sequels. Cyanosis, foaming, and loss of knee jerks are present in the period of unconsciousness; in the stage of clonic spasms the knee jerks are exaggerated and the tongue may be bitten. The sphincters become



relaxed and there is general flaccidity. This may be followed by temporary consciousness and vomiting, or end in stuporous sleep and perhaps intense headache on waking. Subsequent examination may reveal punctate ecchymoses, spots like flea-bites, on the neck and shoulders, injected conjunctivæ, a bitten tongue and bronchi filled with froth. Between the fits there are noted bodily and mental exhaustion, and gradual deterioration in character. The child becomes silly, quarrelsome, spiteful, violent or imbecile.

*Petit Mal* is associated with intractable epilepsy and may precede it by a number of years. Consciousness is lost, impaired or clouded; the patient may respond to orders. Convulsions are absent, or rudimentary and atypical. There may be a momentary pause during speaking; faintness and mental confusion; pallor, fixation of the eyes, dilated pupils and dazed look; an aura with transient unconsciousness; or momentary giddiness with short loss of consciousness during which the child may fall and have slight tonic spasms and enuresis. In rare instances it takes the form of apparently a mere trick, e.g. slow rotation of the head, localised twitchings, and nodding or procursive epilepsy. The pupils are dilated and fixed.

*Procursive Epilepsy* is peculiar to childhood and youth. It causes involuntary movement forward, generally a loud cry, unconsciousness, and seldom an aura. It may last for many years before merging into ordinary epilepsy. It is usually associated with a sclerotic cerebral lesion but may occur without demonstrable alteration in the nerve centres. It is often complicated by moral insanity.

*Ambulatory Epilepsy* is rare in childhood; so too psychic epilepsy, in the form of auræ and maniacal excitement or depression. It may alternate with fits. Automatic actions and a maniacal state may initiate, take the place of, or follow a fit. A girl, aged 4 years, had a mild fit and remained in a very excitable state threatening to "end" herself, cut off her nose, etc. Dreamy mental states may take the place of petit mal or occur in conjunction with epilepsy. They are often merely due to brain exhaustion.

*Jacksonian Epilepsy* depends on a unilateral local cerebral lesion. The spasm begins locally, and may remain limited or terminate in general convulsions. It usually begins in the hand or face. If bilateral, one side is affected first and most. The past history is very important. Most cases are associated with hemiplegia and begin with a severe convulsion during the first 3 years of life, lasting on and off for several hours, or perhaps a single fit. The unilateral character may be present at the onset. The first attack often comes on during an acute fever, whooping cough, the prostration of severe illness, or after injury. Congenital defects of the brain account for a certain number. The initial fit or series of fits may be followed by recurrence for a month or two, or after an interval of a few years. Complete continuity is not very common. The subsequent fits are apparently due to sclerosis. Indications of cerebral palsy may be present in the form of



asymmetry of the face and skull, slight difference in the size of the limbs, possibly a short leg or scoliosis, left-handedness and mental backwardness.

**Diagnosis.**—In true epilepsy the first fit is short and like the succeeding ones. Both convulsions and unconsciousness may be absent. In secondary epilepsy the first fit is often very prolonged and indicative of the toxæmic or infective cerebral disease. Subsequent attacks become shorter and more like true epilepsy. Many fits may follow quickly after the first, whereas in true epilepsy the intervals are prolonged. Secondary epilepsy is little influenced by bromides; and the patients are often excitable and emotional. The blood shows no changes in pure epilepsy; in other forms there may be leucocytosis and eosinophilia at the time of the fits, suggestive of a toxæmic origin.

**Prognosis.**—Epilepsy shortens life. Death occurs from status epilepticus in 25, pulmonary phthisis in 25, and heart disease in 10 per cent., and in the remainder from pneumonia and other lung affections, gastrointestinal derangements and malnutrition. Very few die from asphyxia or injury during a fit. Recovery takes place in less than 10 per cent. It may occur though the fits are very numerous. Thus a boy, 4 years old, had many fits for 9 months, even 273 in 10 days. No cause was found and the fits entirely ceased, leaving no ill effects. Mental enfeeblement and dementia come on eventually in most cases. Petit mal and nocturnal epilepsy are the least amenable to treatment. In view of the grave prognosis and the fact that some cases follow eclampsia, it is of the utmost importance to prevent infantile convulsions and their recurrence.

**Treatment.**—*Of Infantile Convulsions.*—When summoned to a child in a fit the doctor should take with him chloroform, chloral hydrate solution of known strength and, though less necessary, amyl nitrite, solution of morphia and a hypodermic syringe. As soon as the fit starts the child should be laid down, with the head a little raised and the clothes loosened or removed. He must be kept quiet and not disturbed more than necessary. A hot or mustard bath, or a mustard pack, relieves colic and may reduce congestion of the brain by dilating the vessels of the skin. Apply cold to the head at the same time. If there is high fever, cool the bath down to 60° F. The bath should not be given for fits due to syncope or collapse of the lung. Give inhalations of chloroform to control the spasms until other measures can exert their effect.

Empty the lower bowel by an enema of glycerine, salt solution, or sodium sulphate dr. 1-4 in water 5-10 oz. After the bowels have acted, or even before in bad cases, give a rectal injection of chloral hydrate grs. 5-10, according to the age. It can be repeated within an hour, if necessary. Children stand chloral well. If it is not retained, give an injection of morphia gr.  $\frac{1}{30}$ - $\frac{1}{20}$  and atropine gr.  $\frac{1}{200}$ , to a child of  $\frac{1}{2}$ -2 years of age. This may also be repeated in an hour's time. It is safer to rely on chloral and to give chloroform inhalations until the chloral begins to act. If there



is unsuitable food in the stomach, it may be evacuated by lavage or vomiting, induced by tickling the fauces with a feather. Emetics are not advisable. Usually irritant food has passed through the pylorus before the fit occurs. Oxygen gas is useful in cases of cyanosis.

As soon as the child can swallow, give a dose of calomel to empty the intestines and relieve cerebral congestion. Lancing the gums and leeches are useful in cerebral congestion caused by asphyxia. Abundant urination may be looked on as a critical phenomenon announcing the speedy termination of the fit. Active treatment can then be discontinued. For idiopathic multiple fits give chloral grs. 1-2, or more, every 2 hours until the fits have ceased for 24-48 hours and the baby is almost too drowsy to swallow. Great care in feeding is necessary or inhalation pneumonia may be set up. Leave the drug off gradually.

After the attack the child must be kept quiet for a few days, on a light diet, the bowels carefully regulated, and moderate doses of bromide given. During sleep the room must be well ventilated, the feet warm and the head raised. All sources of reflex irritation should be attended to, and any disease present efficiently treated. On account of the frequent presence of rickets cod-liver oil, malt and iron are the most useful drugs for the prevention of infantile convulsions. With improved nutrition the central nervous system becomes less liable to explosive disturbance.

*Treatment of Epilepsy.*—Insane, feeble-minded or imbecile epileptics should be kept in asylums or epileptic colonies. An intelligent epileptic can be brought up at home in the country, and live the life of a healthy animal. Regular sleep, routine life, open air and exercise are essential. An efficient and valuable education can be obtained from the objects of country life. School-life is permissible, if the fits are mild and infrequent and the child mentally sound. Over-exertion, mental and physical, worry and excitement must be avoided. Boys, who have to earn their living, should be brought up to country pursuits or some outdoor occupation, involving more or less physical labour and no dangerous positions or exposure to great heat. Careful attention must be paid to the intestinal tract, constipation remedied, and intestinal antiseptics given if necessary. The diet should be plain, unseasoned, limited in amount, and contain little meat and little salt. Bread can be made with bromide of soda (Bromopan). Alcohol, tea, coffee, meat extracts and other stimulants must be avoided. As antiseptics give borax and  $\beta$ -naphthol grs. 15 daily, or salol grs. 10 in 5 oz. of water, night and morning. Many fits are toxic in origin and can be prevented by an occasional blue pill and black draught.

Bromides, up to drs. 3 daily, given night and morning, and combined with salt starvation, should be continued for 2 years in bad cases. The addition of opium is sometimes useful. Borax, belladonna, salts of zinc, and valerianates are the next best drugs. Pot. iod. is sometimes useful. For fits on rising, give a light meal and medicine an hour beforehand.



Treat nocturnal fits with bromide and borax, and seek for asphyxial causes. Or give a nightly dose of bromide, nux vomica and digitalis. Give valerian if there is any hysteria; belladonna for vasomotor auræ; bromides for sensory auræ; morphia for motor auræ; and cannabis indica for psychical auræ and persistent headache. Of the bromides the strontium salt is the least likely to cause rashes. Bromide is particularly useful in combination with phenazone. Nux vomica can be added to prevent depression, digitalis if the heart is weak, and citrate of iron and ammonia if there is anæmia. Urethane may be efficacious when bromides fail. Trephining sometimes relieves Jacksonian epilepsy.

The *status epilepticus* may be induced by constipation, feverish attacks, insolation, alcohol or the sudden withdrawal of bromide. Death results from asphyxia, due to prolonged respiratory spasm or entrance of food into the larynx, septic pneumonia or intracranial hæmorrhage. It is treated by large doses of chloral per rectum, or tr. opii with an equal quantity of tr. strophanthi if the heart is weak; chloroform inhalations, with oxygen if there is asphyxia; a cold pack, if there is high fever; or hyoscine subcutem. Venesection is rarely needed.

**Tetany.**—*Syn.*: *Tetanilla*.—This was first described by Steinheim of Altona in 1830, and named “*tetanie*” by Corvisart in 1852. It is a spasm of the muscles which especially affects the extremities, notably the hands and feet. The spasm is tonic in character; continuous, intermittent or paroxysmal; almost invariably symmetrical, though occasionally limited to one limb. It is of uncertain duration, persists during sleep, and is not associated with loss of consciousness or impaired intelligence. It is not due to organic nerve lesions, though it has been recorded in connection with cerebellar tumour.

*Etiology.*—The geographical distribution varies with climatic conditions and the amount of attention devoted to childish ailments. It is most frequent between November and April. Cold is a predisposing and an exciting factor. Griffith found only 50 cases, 38 in children, reported in America (1895). Family predisposition may be disregarded. In some families the children seem more liable than in others. I have known it occur in 2 brothers.

It is infrequent before the fourth month and after the third year, and most common in the first 2 years of life, especially from 6-18 months, when rickets is prevalent and children are often badly fed. Out of 14 successive cases 7 were in the first, 5 in the second, and 2 in the third year of life; 10 of them were males. During the early years of life males are much more susceptible but at a later age the females preponderate.

Rickets is present in quite half the cases but tetany can occur with little or no evidence of it. Gastric and enteric disorders are common exciting causes, probably through toxæmia, though occasionally reflexly as in painful pyloric spasm. Tetany occurs in dilatation of the stomach in adults,



and in infants may be due to dilatation from overfeeding, rickets, febrile diseases, pneumonia, and congenital hypertrophy of the pylorus. A dilated stomach is a chronic disorder. It is the chronic alimentary disturbances which are associated with tetany, rather than acute attacks in which toxins are rapidly eliminated. It has been reported in cases of dilated colon; disappearing after irrigation but reappearing on accumulation of the fæcal material.

*Pathology.*—It is due to hyper-excitability of the nerve cells in the bulb and anterior cornua, of the nerves and of the muscles, due to toxæmia. Nutritional and functional damage of the nerve cells in the cord is shown by the muscular weakness and atrophy sometimes present. The electrical reactions indicate that the upper neuron is not affected by the toxin. Rickets may interfere with its development and power of control over the lower neuron. The cranial nerves are sometimes affected; facial irritability is common and squint may develop. Bilateral symmetry and the uniform character of the spasm indicate a spinal affection. Irregular distribution is due to special susceptibility of certain nerve cells or unequal dissemination of the poison.

The poison is derived from the products of bacterial activity or imperfect digestion. Toxic bodies have been isolated from the stomach but so far tetany has not been produced experimentally. It is curious that tetany may be induced or exaggerated by lavage of the stomach in cases of congenital pyloric stenosis. Stoeltzner suggests that tetany is due to excess of lime salts, which are 5 times as abundant in cow's milk as in human milk. He states that the addition of chloride or acetate of lime to the food exaggerates the symptoms. The association of rickets he explains as due to excess of lime salts in the blood, because of deficient bone formation. On the other hand it has been ascribed to deficiency of lime. Silvestre states that the brain contains less lime than normal. There is no reliable evidence that any component part of pure milk is the primary cause. Only one of my cases was breast-fed. The others were having a diet of cow's milk or condensed milk, and usually other articles of food sufficient to cause intestinal derangement. Kussmaul's theory that it is due to dehydration of the body, especially the muscles and nerves, can hardly be said to hold water. Its relationship to the parathyroids is considered on p. 208. The most probable explanation is that it is due to an intestinal toxin acting on nerve cells whose nutrition is impaired by rickets or other causes.

*Symptoms.*—The spasm may come on quite suddenly, perhaps during an attack of laryngospasm or general convulsions. Usually it develops slowly, in the course of some alimentary complaint or during convalescence. Thus a boy, aged 14 weeks, had hæmorrhagic oedema, probably angioneurotic in origin, and secondary colitis. When convalescent and eating well he developed typical tetany which lasted 3 days. Older children may complain of premonitory symptoms such as headache, malaise, and burning, tingling, numbness or formication.



The spasm is sometimes only present on waking from sleep. It begins at the periphery and spreads upward. The hands are first affected, and assume the position known as "the gynæcological or obstetrical hand" or "the hand of the accoucheur." The fingers are in the attitude of holding a pen, partially flexed at the metacarpo-phalangeal and fully extended at the other joints, with their tips approximated. The thumb is fully extended and adducted, its tip applied to the palmar surface of the terminal phalanx of the first or second finger; or it may be flexed across the palm. The inner and outer borders of the palms are approximated, hollowing the palm. The wrists are immobile, flexed, tense and shiny, perhaps red, and drawn towards the ulnar side. The forearm is semi-pronated and flexed at the elbow, and the arm is adducted. The feet are rigid in the position of equino-varus, with inversion of the sole and approximation of its inner and outer borders. The sole is contracted longitudinally and becomes markedly concave, with a median furrow. The toes are extremely flexed and overlap, or are flexed at the metatarso-phalangeal and extended at the other joints; or the big toe is hyperextended and the other toes flexed. The dorsum of the foot is tense, shiny, and perhaps red. Both hands and feet may be a little swollen with œdema, and this swelling may be the first sign.

Commonly the hands alone are affected. Next in frequency come the feet. Other muscles of the limbs may be involved. The legs and thighs are sometimes flexed; or the adductors may be so strongly contracted as to cause crossing of the legs. Spasm of the neck and back muscle produces retraction of the head, rigidity of the neck and opisthotonos. Dyspnœa may result from spasm of the thoracic muscles. Wrinkled brow, pouting lips, squint, trismus and risus sardonicus are sometimes present. Difficulty in swallowing and pupillary rigidity have been noted. The abdominal muscles, bladder, diaphragm and indeed every muscle of the body may be involved. In 2 cases I have seen marked tremor of the head and neck, and in 1 of them the hands and arms were similarly affected. Paræsthesiæ and tenderness in the limbs may be present. Pain, due to the cramp-like spasms or to passive extension of the muscles, induces sudden cries.

Tetany does not cause fever. If present it is due to the intestinal condition or some complication. Variable appetite, more or less vomiting, and loose undigested stools are almost constant. In only one case were the stools normal. The knee jerks are variable. Albuminuria is not infrequent. Drowsiness or semi-coma may be present and a sign of toxæmia. The special signs known as Trousseau's phenomenon, Chvostek's sign and Erb's sign (p. 617) are often present. *Carpopedal spasm* or "Arthrogryposis," is a variety in which the fist is clenched, with the thumb in-turned and flexed and the fingers flexed over it. The toes are flexed and the big toe sometimes abducted and hyperextended and the heel drawn up. This variety of spasm is seen in other affections, notably eclampsia. In another type the thumbs



are similarly flexed and in-turned, and the fingers flexed at the metacarpophalangeal joints and hyperextended at the other joints.

The chief *complications* are laryngospasm, convulsions, enteritis, bronchitis and broncho-pneumonia.

The *diagnosis* is based on the character of the spasm. "Latent" tetany is associated with rickets, alimentary disturbance, laryngospasm, convulsions and the various accessory phenomena. Occasionally the spasm is typical in one attack and carpopedal in subsequent attacks. The latter type of spasm may occur in cerebral sclerosis, sometimes with fine tremors and convulsive movements of the arm.

*Pseudo-tetanus* occupies an intermediate position between tetany and tetanus. Escherich has described under the name of "pseudo-tetanus of the newborn" a condition of opisthotonos and recurrent arrest of breathing. It is sometimes called "acute recurring respiratory failure." Pseudo-tetanus has been recorded in a few children between 4 and 10 years of age, nearly all boys. The muscles of the jaw, neck, back and legs, are affected by persistent chronic spasm; the masseters and muscles of the back being chiefly involved. In this respect it resembles tetanus, and probably many cases of idiopathic tetanus ending in recovery are of this type. It is usually abrupt in onset but the symptoms quickly assume a milder type. Stiffness of the legs, rigidity of the jaws, trismus, opisthotonos and rigidity of the extremities are present. The muscles of the trunk, neck and legs are powerfully contracted and stand out prominently, as hard as marble or rigid as wood. Acute muscular spasms are mild, infrequent, and may be induced by cold, noise, handling and emotion. The arms, hands and eyes are usually unaffected. Knee jerks are active. Relaxation is incomplete while at rest and asleep. Violent attacks of laryngospasm and cyanosis, diaphragmatic spasm and dyspnoea may occur. The patient is not emotional or nervous; remains cheerful and comfortable in spite of the unnatural attitude; and recovers gradually in from 1-6 weeks. There is no spasmophilic over-excitability and tetanus antitoxin is of no value. Chloral, bromides and morphia are the most useful drugs.

*Prognosis.*—Tetany varies in duration and severity. It may last for hours or persist for months, usually only 2 or 3 days. It may be mild, painless and continuous, or severe and paroxysmal. In infants it is usually of the first type and present while asleep. Paroxysms may last a few minutes or for 24 hours. The intermittent or paroxysmal type is the one most often seen in the rachitic. It runs an acute or subacute course with short and painful cramps, increased mechanical and electrical reactions, and a tendency to recur on slight provocation. One child had an attack in her third year, which persisted on and off for 3 months, and 3 more attacks in the next 4 years. The disease is not fatal in itself and the prognosis is that of the cause. It is unfavourable in marasmus, prolonged diarrhoea, bronchitis, broncho-pneumonia, and œdema of the lungs. Tremor and attacks of



dyspnœa are bad signs. Drowsiness and semi-coma indicate severe toxæmia. There is always the liability to death from laryngospasm or convulsions and a tendency to recurrence.

*Treatment.*—It can be prevented by attention to the general health, warmth, proper clothing and diet. When present, the underlying cause must be treated. For alimentary troubles give calomel, alkalies, HCl., bismuth, benzo-naphthol, etc.; and diet the child carefully. Chloral and hot baths are the most efficacious methods of relieving the spasm, and bromides are of some value. Rest in bed, dark room and complete quiet are necessary in bad cases. Treat the rachitic condition when the attack is mild, the digestion good, and the patient convalescent. Lavage and irrigation of the colon are occasionally required.

**Laryngospasm.**—*Syn.:* *Laryngismus Stridulus*—*Spasmodic Croup*—*Spurious or False Croup*—*Cerebral Croup*—*Child Crowing*—*Inward Convulsions*—*Glottic Spasm*—*Holding Breath Spasms*.—This is a form of local convulsion, characterised by spasmodic closure of the glottis, due to spasm of the adductor muscles or inco-ordination of the muscles of the larynx. In severe cases other muscles of respiration and of deglutition are involved. The typical symptom is a peculiar crowing sound on inspiration, following a period of apnœa. Occasionally the glottis escapes, and the spasm is limited to the diaphragm and external muscles of expiration; in these there is no crowing. The crow is due to inspiration through a narrow glottis.

*Etiology.*—The affection is most common in families with a neurotic taint. Males are said to be twice as liable as females, but in my experience they only slightly preponderate, in the proportion of 5-4. It is most frequent between 6 and 18 months of age. It is decidedly uncommon after the first dentition. About 90 per cent. of the cases occur before the milk teeth are fully erupted. The attacks are most common in the first 5 months of the year, at any rate in London. Barlow, from observations on Manchester children, found that 70 out of 114 cases occurred from October to February. The connection with rickets is the most marked etiological factor, for this disease is almost invariably present. It is frequently associated with tetany also and usually presents the signs of spasmophilia. Some cases occur in healthy infants in the first 2 weeks of life, and appear due to adenoids or to post-nasal catarrh grafted on adenoid hyperplasia; the frequency and severity of the attacks varying directly as the degree of nasal discomfort. Exciting causes are a draught of cold air while asleep, tickling the throat or external ear, an elongated uvula, any interference with normal respiration, overdistension of the stomach, laughter and emotion. Thus the predisposing causes are rickets and increased excitability of the nervous system, and reflex irritation, alimentary or pharyngeal, is the chief exciting cause.



*Pathology.*—It has been ascribed to craniotabes, through pressure on the softened occiput. Occasionally attacks can be produced by pressure on the softened parts, but on the other hand many occur when the child is sitting and when there is no craniotabes. There is no evidence that it is due to pressure of an enlarged thymus or mediastinal gland. There is distinct evidence that catarrhal conditions of the pharynx or larynx, and that the entrance of saliva, mucus or milk into the glottis may produce an attack. Most probably it is due to the action of deficiently aërated blood on the respiratory centre, and for this reason the attacks are most common at night in children continually breathing impure air. The important facts to bear in mind are the connection with rickets and craniotabes; the general malnutrition often present; the malnutrition of the blood and nerve centres; the liability of the nervous system in infants to spasmodic affections; and the connection of other nervous phenomena with the rachitic condition. In fatal cases there is no constant lesion. The epiglottis is sometimes impacted and the mucous membrane of the larynx congested.

*Symptoms.*—Usually the child is supposed to be in perfect health, but sometimes there is an antecedent history of fretfulness and irritability. The attack comes on at night and awakens the child from sleep. It may occur in the daytime, during sleep or even when awake. It may come on while suckling and recur at each attempt to nurse. In a typical case the child awakes suddenly about midnight, makes a few attempts at respiration, a long crowing or whistling inspiration, and then the sounds cease from complete closure of the glottis. At first he is pale or slightly flushed. The face has an anxious expression and gradually becomes blue or livid, especially about the lips. The eyes are wide-open and staring, or rolled upward and outward. The head is thrown back, the body rigid, and in severe cases there are clonic facial spasms, cyanosis and marked opisthotonos. *Carpo-pedal contractions* are often present. If old enough the child sits up in the cot, is restless, throws itself about from side to side, and is evidently very frightened. The pulse is small, perhaps almost imperceptible. The fontanelle bulges from venous engorgement. After the spasm of the glottis has lasted for a few seconds, it ends suddenly with relaxation and a long crowing inspiration. The child then bursts out crying, vomits or has a fit of coughing, and falls asleep from exhaustion. In mild attacks the child rolls up the eyes, crows, may flex the thumbs in the palms and recovers in a few moments. It then bursts out crying or has a fit of temper. The attack may be mistaken for a mere exhibition of temper, if the symptoms of the latter are well marked while the attack is merely momentary.

In very young infants the attack may come on silently with no preliminary crow. The spasm affects the diaphragm and respiratory muscles, except the glottis. The body may remain limp, motionless and without



respiratory effort, while the face grows darker and darker for a few seconds ; or there may be a general rigidity with in-turned thumbs.

The duration is variable, usually a few seconds ; and even in attacks in which the child appears on the verge of dissolution it probably does not last more than a minute. Occasionally the spasms recur so rapidly that for many hours the child passes from one into another, a condition like that of status eclampticus. Usually the child seems quite well after an attack and drops off to sleep. Next day it seems in perfect health, but at night, often exactly at the same time, the same cycle of events is repeated. There may be only one attack, or recurrence may take place for months whenever an exciting cause is present. Several paroxysms may occur in the same night, or even hourly by night and day, and occasionally more frequently.

*Diagnosis.*—The sudden onset, especially if at night, the characteristic crow, carpopedal contractions, rickets, good health between the attacks, and the absence of fever or constitutional disturbance are sufficient to base a diagnosis upon. Catarrhal affections of the larynx, retro-pharyngeal abscess and congenital laryngeal stridor must be excluded. “Holding-breath” may be due to mere anger. Syncopal attacks without crowing may be mistaken for minor epilepsy. The glottic spasm occasionally occurs in organic brain disease.

*Prognosis.*—The outlook is favourable under suitable treatment but there is always the liability to recurrence and to death in even a mild attack. As a rule the more frequent the attacks the milder they are in character. There is no subsequent hoarseness, fever, or respiratory trouble. It is a cause of sudden death in apparently healthy infants. A fatal result may be due to the epiglottis becoming wedged in the chink of the glottis and not released when the spasm relaxes, or may result from expiratory apnoea due to severe spasm of the glottis, diaphragm and respiratory muscles. Should a spasm pass into general convulsions and unconsciousness the child is more likely to die. The usual causes of death are suffocation, asthenia, repeated attacks, pulmonary collapse, deglutition spasm and starvation.

*Treatment.*—Mild attacks require no treatment as they are merely momentary. In more severe ones the child should be raised to a sitting posture and may be carried about, but should not be allowed to lie down. The room must be well ventilated and no undue crowding about the patient permitted. A sponge or flannel wrung out in hot water and applied to the throat may abort the attack. For immediate relief hook the epiglottis forward with the finger ; stimulate the conjunctiva by touching it with the finger ; give inhalations of chloroform, ether, amyl nitrite, smelling salts or acetic acid ; apply ice to the larynx or epigastrium ; slap the face or chest with the end of a towel dipped in cold water or pour cold water on the head with the child in a hot bath. Vomiting may be induced by tickling the



fauces with a feather or inserting the finger. If the epiglottis becomes impacted it must be at once released by the finger. Artificial respiration should be taught to the attendant in a bad case and should be adopted for sudden pallor, dilatation of the pupils, and cessation of struggling and breathing.

To prevent recurrence treat the underlying rachitic condition and give bromide in doses of grs. 4-10 at night, or in smaller doses more frequently if the attacks occur by day. Phenazone at bedtime is also beneficial. Chloral, trinitrin and opiates are sometimes used, but it is rarely necessary to have recourse to any drugs except bromides and cod-liver oil. It is of importance to regulate the child's diet and to insist on the hygienic measures appropriate in rickets ; to treat post-nasal catarrh and to remove adenoids if present ; to avoid over-feeding, keep the bowels open, and insist on a plentiful supply of fresh air by day and night.

**Myoclonus.**—*Syn.:* *Myokymia* — *Paramyoclonus Multiplex* — *Myoclonus Epilepticus*.—Children of a neuropathic or alcoholic heredity, at 5-15 years of age, are sometimes affected with sudden shock-like contractions of a muscle bundle or a whole muscle, causing fibrillary twitching or sudden movement of a limb. It never affects groups of muscles and is never unilateral, being generally symmetrical and often isochronous. Its effect is analogous to that caused by a single induction shock. The movements are not rhythmical and not like voluntary ones. They last for several minutes. The ocular muscles are never attacked, the sphincters are unaffected, and the face often escapes. The proximal segments of the limbs are usually the most involved. The superficial reflexes are increased and the deep ones are variable. There is no atrophy, no change in electrical reactions, and no abnormality of sensation. The movements may interfere with volitional movements, cause inability to dress, and affect speech. They generally cease during sleep. Slight spasticity may exist in long-standing cases. The affection is slowly progressive for a time and then stationary for years. Its intractability varies with its duration. A few cases are fatal within a few months ; some recover, but are liable to recurrence. Epileptic attacks are a frequent association. Death is usually due to intercurrent disease.

No explanation has been found. Probably there is some mischief in the motor region, for it is associated with epilepsy and ceases during sleep. Myoclonus may occur in sclerosis of this region. The diagnosis depends upon the simple shock-like movements in symmetrical muscles on opposite sides, bearing no resemblance to voluntary movements and destitute of rhythm. In hysteria the movements are less persistent and there are other functional derangements. In gross cerebral disease there are signs of cranial mischief. Treatment consists in maintenance of general nutrition, arsenic and hydrotherapy.



*Chorea Electrica of Dubini* (1849).—Children of either sex, age 9-15, are affected with sudden muscular contractions of the neck and shoulder muscles. The cases described under this name are frequently simple habit spasm or habit spasm in a hysterical subject. A few are associated with petit mal or Jacksonian epilepsy, and due to a grave lesion of the cortex; others are identical with certain cases of myoclonus. In the disease described by Dubini there is paresis, atrophy, loss of faradic irritability, and convulsions which may be unilateral. There may be high fever in acute cases. It is progressive and often fatal. No constant morbid change is found.

**Habit Spasm.**—*Syn.:* *Tic—Co-ordinated Tic—Tic Convulsif—Habit Chorea—Mimic or Histrionic Spasm.*—A tic is a co-ordinated purposive act of voluntary type, provoked in the first instance by an external cause or an idea; repetition leads to it becoming habitual, and finally to its involuntary reproduction without cause and for no purpose, at the same time as its form, intensity and frequency are exaggerated. It thus assumes the characters of a convulsive movement, inopportune and excessive; its execution is often preceded by an irresistible impulse, resisted at first and then overwhelming and followed by misery at giving way to it, its suppression associated with malaise. The effect of distraction or of volitional effort is to diminish its activity; in sleep it disappears. It occurs in predisposed individuals, who usually show other indications of mental instability. Such is the description, slightly modified, given by Meigs and Feindel (1908).

“Habit spasms” is not a good name, for they are not true habits and a spasm cannot be controlled by an act of will. Guthrie speaks of them as motor manifestations of psychical unrest or cortical instability. In a way they are an emotion of expression; expressive gestures, preceded by a desire to perform them and followed by a feeling of satisfaction. The expression varies from a single twitch of a single muscle to complicated co-ordinated movement.

*Simple Tic.*—The movement is usually only of one kind, stereotyped, and commonly involves the muscles of the face, especially the eyes and mouth, and those of the head and shoulders. The lower limbs are much more rarely affected. As a rule it is unilateral and recurs every few minutes, occasionally with great rapidity one after another. It differs from chorea in the same movement being constantly repeated. Sometimes the recurrent motions are not identical. They are restrained and much more limited in extent while under observation, but worse afterwards, more or less controllable, increased by excitement, and cease during sleep.

*Varieties.*—There are numerous varieties, such as blepharospasm, nictitating or ocular spasm, due to spasmodic contraction of the orbicularis muscle and producing winking, blinking, or screwing up the eyes; sometimes extending to other muscles of the face or involving the eyebrows and occipito-frontalis muscle. It is most common in school children and may



take its origin in hypermetropia or hypermetropic astigmatism, blepharitis, fissure at the angle of the lids, ingrowing hair, and affections of the conjunctiva or cornea. If due to a phlyctenule or keratitis the spasm is generally tonic in character. A true spasmodic squint is occasionally seen. In facial spasm most of the muscles of one side of the face are involved or only the zygomatics, drawing up the angle of the mouth on the affected side. Other varieties are shrugging of one or both shoulders, tossing up the chin, lateral movements of the mouth, elevation of the upper lip, showing the teeth or snarling, nose twitching, nose picking, sniffing, sucking, licking, yawning, biting, masticating, grunting, barking, stamping the feet, jerking, hopping and leaping; occasionally echolalia and coprolalia.

*Etiology.*—It is more common in girls than boys, and in neurotic children or those of neurotic parentage. The patients range from 3 years to puberty, usually 5-9 years. They are generally thin, nervous, excitable children of good intelligence; and often present other signs of nerve instability, e.g. timidity, somniloquence, pavor, somnambulism, headaches, enuresis, debility and undue emotionalism. It is perhaps a symptom of psychasthenia. Bad health and bad hygienic surroundings are predisposing factors. The chief exciting causes are mental excitement and the effects of education, such as strain, competition, dread of failure, fear of punishment, teasing and bullying. A few cases follow fright or surgical shock. The particular movement may depend upon some peripheral exciting cause, which suggests rather than produces it. It is then maintained because of the neuropathic state. Sometimes it is a pure habit or family peculiarity, largely dependent on imitation and suggestion. Such a habit may be transmitted from an unknown grandparent. Mimic or histrionic spasm is a name which should be reserved for epidemics of any particular type in several children of the same family or at the same school.

The spasm may persist throughout life. The prognosis varies with the age of the child and duration of the spasm. Usually it subsides gradually under treatment, if not too much attention is paid to it. Frequently the type changes, as one spasm goes another comes.

*Convulsive tic* is simple tic in a severe form and occasionally combined with explosive utterances. It is rather more common in males and usually begins at 10-15 years of age. The spasm is less restricted to a simple movement and may be widely distributed. It causes grimaces, pantomiming, grotesque attitudes and gestures, and sometimes imitation of gestures (Echokinesis).

*Psychical tic* or *Gilles de la Tourette's disease* is a further stage of convulsive tic, with no motor manifestations. It is far the most serious variety, and possibly due to organic cerebral mischief. In the exclamatory tics there are explosive utterances, articulate or inarticulate, which may take the form of echolalia or repetition of words and phrases, coprolalia or the use of obscene words and bad language, or the cry of some animal.



In another form there are fixed ideas, the *folie de doute* and various phobias. Some cases are characterised by impulsive acts and in others there is general mental impairment. The disease usually begins at 10-15 years of age, runs a progressive course with intermissions, and often ends in insanity. Recovery is rare.

*The Diagnosis.*—Simple habit spasm must be differentiated from the stereotyped acts; various spasms, e.g. of the face, trismus, spasmus nutans and torticollis; chorea, tremors, myoclonus and athetosis. Hysterical spasm is worse under observation and may be cured suddenly. It is doubtful whether one is justified in classifying Simple Tic with Psychological Tic and regarding Convulsive Tic as an intermediate stage, although apparently it is a close connecting link. Yet on the one hand simple tic is harmless, temporary, unimportant and a common affection; whereas psychological tic is rare and of very grave prognosis.

*Treatment.*—Very little notice should be taken of the habit and it is essential to avoid scolding and punishment. A system of encouragement and rewards may assist in strengthening the will power and diminishing the recurrence. Any peripheral exciting cause present should be cured. For instance zygomatic spasm, in a 6-year old boy, subsided as soon as a decayed molar tooth on the same side was removed. In blepharospasm remedy any defect of vision. Unsuitable spectacles may give rise to spasm, which becomes permanent unless the use of them is stopped. In mild cases school life is not contra-indicated, provided the child gets long hours of sleep, is not teased or bullied, and is not overworked. Frequently a change to the country or seaside is rapidly curative. A certain amount of benefit is derived from movements directed to preserving immobility and replacing incorrect ones by normal ones. For this purpose the selected exercises in gymnastics should be done under supervision and in front of a long mirror. It is rarely necessary to do more than prevent eye-strain and overwork; insist on good general hygiene, fresh air, exercise and liberal diet; and give tonics such as arsenic, cod-liver oil, iron, quinine and strychnia. Bromides and valerianates are often useful. Psychological tic must be treated as a nervous disease liable to end in insanity. This involves peace and quiet, no emotional excitement, no mental strain, and a simple country life.

**Nodding Spasm.**—*Syn.:* *Spasmus Nutans*—*Head Nodding, Jerking or Shaking*—*Eclampsia Nutans or Rotans*—*Gyrospasm.*—This affection is characterised by movements of the head on the trunk, commonly associated with nystagmus. The movements vary somewhat in different cases. Usually they take the form of rotatory nodding, as if due to the spasmodic contraction of one of the sterno-mastoid muscles. In my experience the right muscle is most often involved, causing the head to be drawn downward to the right shoulder and the chin to be pointed toward the left. Occasionally the movements are pure nodding ones, like those of a mandarin doll, and due to simultaneous spasm of both sterno-mastoids. This variety is



most frequent in microcephalus. Sometimes the head is simply moved from side to side.

The movements are not constant, but may be remarkably frequent and almost continuous. They are increased by excitement and by fixation of the eyes, as on looking at a bright light. They cease during sleep and recumbency, and may disappear when the eyes are covered, or if one eye alone is covered when the nystagmus is limited to that eye. Neither face, trunk nor extremities are involved. In about half the cases the head is held in a peculiar way, cocked on one side, thrown backward and less often forward. The child sometimes looks at objects out of the corners of the eyes.

Nystagmus is generally present in one or both eyes, and much more in one than the other. The movements are very rapid, more so than those of the head, and best marked when the eyes are fixed or the head is held. They are usually lateral, 4-6 a second and of short range, quivering, twittering or rolling. Generally they are horizontal or combined with some rotation. They are not constant and may be present apart from the head nodding. Sometimes they are only induced by fixation of the eyes, by restraining the movements of the head, placing the child on its back, or making the child look to one side. On conjugate deviation they may not be present when the deviation takes place to one side and well marked when it takes place to the other. The movements of the head and eyes are often, but not always, in the same direction. Convulsive movements of the lids are not infrequent and the upper ones may be markedly retracted. Rarely there is spasmodic convergent or divergent squint.

The disease is exceptional in the summer and is most common in winter and spring, possibly from want of light through living in dark rooms. Either the head nodding or nystagmus may begin first; more usually they are simultaneous in onset. In one of Gee's cases nystagmus began at 4 weeks and the head movements at 6 weeks. The usual age is 5-12 months, but it may begin at 18 months. Males and females are equally liable, and more than one child in a family may be affected. There is no definite connection with rickets though it is frequently present. Craniotabes is almost always absent. Dentition cannot be regarded as a cause though it may aggravate the condition. The movements may appear before dentition begins. These babies are usually precocious, of more than common intelligence at the age of onset, and good-tempered. Some of them are liable to attacks of unconsciousness or possibly vertigo.

The combination of head jerking and nystagmus suggests that the nuclei of the spinal accessory and upper spinal nerves are in close relation to those of the oculo-motor nerves. The movements are rarely confined to the sterno-mastoids and often affect the muscles which rotate the head. In many the rotatory movements are the most marked. Apparently it is a derangement of co-ordination. Normally the movements of the head and eyes are not harmoniously associated until about the fourth month. In



most of these cases the patient takes notice early, develops precocious movements, and is liable to derangement of such movements. If this is true, the defective co-ordination may be due to overstraining the eyes to look in a particular direction, aided by nutritional weakness. In some respects it is analogous to miner's nystagmus.

It must be *diagnosed* from congenital or juvenile nystagmus due to amblyopia, a central macula or cataract; the swaying and rolling movements in imbeciles; and from eclampsia nutans, a form of epilepsy in which there is a series of rapid nods or jerks of the head, with vacant look or momentary unconsciousness. In salaam fits, a sub-variety of eclampsia nutans, the hands are placed in front of the face, palms outwards, and the head and trunk are suddenly bowed down.

The *prognosis* is good. All get well and there is no liability to any nervous disease; if such is present, it is a coincidence rather than a sequel. Many recover in a few weeks to a few months, and it rarely lasts more than 2 years. Relapses are common. The nystagmus persists longer than the spasm.

*Treatment*.—Attend to the general health. The movements are harmless and unimportant. No special drugs are required. Phenazone gr. 1, 2-4 times a day, may do good.

**Head Banging** and other movements such as *head rolling*, *head burying* and *body rocking*, occupy an intermediate position between the spasmodic nervous affections and simple bad habits, for instance thumb biting, pica and masturbation. For some inexplicable reason or from mere temper the child, generally an infant, beats the head with its fists or bangs it against structures, whether hard or soft, such as the pillows or rails of the cot, the walls or the floor. Head banging may alternate with some of the other movements. From a medico-legal point of view the affection is important, as the banging may be severe enough to cause abrasions and bruises.

Most often it occurs as the child is going to sleep but it may take place at any time of the day or night. It resembles epilepsy in the tendency of the child to turn on the face before the attack begins. Sometimes it goes on for several hours or even all night, the face and forehead being banged into the pillow while the child remains fast asleep. In a 6-year old boy with acute nephritis the habit took the form of wrapping the head up in a sheet or blanket and burying it in the pillow or bedding (*head burying*). He was dull, lethargic, with drawling speech, and decidedly backward. Double otorrhea was present.

In *head rolling* the head is rolled from side to side and the hair gets rubbed off the occiput. The movements may continue in sleep and are painless. These children are almost invariably under 2 years of age, often rachitic, and frequently have middle ear trouble. Sometimes the attack seems due to dentition.



*Body rocking* is antero-posterior and rarely lateral. It takes place in a sitting posture and therefore only in infants who can sit up, and may persist in sleep. It is most common in the blind and mentally defective, and is an indication of an eccentric or neurotic temperament, or of imbecility.

All these affections have been found associated with rickets, ear trouble, phimosis, adherent prepuce, masturbation, fits, tuberculosis, and backwardness or actual imbecility. They have no connection with nightmare, pavor or somnambulism. Head banging must not be confused with a mere outbreak of temper in an excitable or neurotic child. It generally begins before the end of the first dentition and may persist for years. It has occurred with intracranial tumour.

*Treatment* often fails. Attention should be paid to the digestion, the general condition of the child and the mental state. The ears must be examined and local sources of irritation treated. Bromides and phenazone are sometimes useful.

**Nystagmus.**—The involuntary movements of the eyeball, known as nystagmus, assume many forms. It is both a symptom and an entity. As a family peculiarity it has been recorded in 2 and 3 generations and may be associated with head nodding. Sometimes it is due to albinism. These children have light, almost white hair and eyelashes, pink pupils and iris, and photophobia. It has been described by Nettleship as occurring in partial albinism with imperfect sight; a condition apt to run in families, affect males and be transmitted by females. It may be secondary to cerebral mischief, e.g. basal meningitis and hydrocephalus; or due to mental defects and cranial peculiarities, as in microcephalus and oxycephalus. Frequently it results from visual defects such as corneal opacities, congenital cataract, choroiditis or high myopia. The movement is generally horizontal, sometimes vertical or rotatory, and occasionally spasmodic, the eyes suddenly converging or diverging.



## CHAPTER LVII.

### NON-SPASMODIC FUNCTIONAL NEUROSES.

*Headache—Migraine—Disorders of Sleep—Nightmare—Somnambulism—  
Pavor Nocturnus—Pavor Diurnus.*

**Headache.**—As a symptom headache occurs in most febrile conditions, toxæmias, constitutional states, hygienic errors, nervous disorders and various physical defects. It is not always possible to ascertain the exact cause and often more than one is implicated. The words of Juncker still remain true “*De doloribus capitis ; scandalo medicorum, difficulter removendo.*” Headache should never be neglected in young children. It is more significant in them than in older people and may indicate the onset of acute or dangerous disease. It is acute or chronic. Sometimes it takes the form of “paroxysmal recurrent headache” or of “migraine.” The former type is not uncommon in children. Bilateral periodical headaches cannot be differentiated from those hemicranial in type. *Dolor capitis*, pain due to changes not inside the cranium, that is in the scalp or bones, must be excluded.

**Etiology.**—The chief predisposing causes are inherited weakness or instability of the nervous system, the gouty and, possibly, the rheumatic diathesis. Children of neurotic parentage, or themselves neurotic, are liable to headache on slight provocation such as excitement, emotional disturbance, and active brain work.

The exciting causes are impaired general health and anæmia, leading to malnutrition of the nerve cells ; alimentary disorders, through the absorption of toxic products ; deficient aëration of the blood, toxæmia, uræmia, and the toxic effects of alcohol, lead and other metallic poisons ; eye-strain, mental and physical overstrain, and excitement or emotion. The headache caused by a vitiated atmosphere is due to excess of CO<sub>2</sub>, deficiency of oxygen, and the absorption of deleterious products given off from the lungs, skin, clothes, etc., of others present. Deficient oxygenation accounts for headache in children with adenoids, congestion of the nasal mucosa and respiratory affections.

The chief organic causes are meningitis, cerebral tumour and abscess, sunstroke, injury, and syphilis. It is often difficult to get a history of injury as a nurse will rarely confess that she has dropped the child.



Provided organic disease can be excluded, it will be found that toxæmic causes predominate, chiefly those dependent on the digestive system.

*Classification.*—Headaches may be regarded as structural, hæmic or nervous in origin. In the structural or organic headaches there is usually increased intracranial pressure and the pain is liable to keep the patient awake. The hæmic causes include anæmia, hyperæmia, fever, and all causes of toxæmia and auto-intoxication. Nervous headache may be due to brain exhaustion and noise; reflex, usually through branches of the fifth nerve, e.g. from decayed or impacted teeth, errors of refraction, otitis, and naso-pharyngeal affections; or idiopathic, e.g. migraine.

*Symptoms.*—In the very young there is dislike of light and noise, constant putting of the hand to the head, supporting the head with the hands, and screaming paroxysmally or when the head is moved. The intensity of the pain varies as the capacity for sensation rather than with the primary cause. It is greater when the fontanelles are closed.

*Situation and Characters.*—Localisation is a doubtful clue to pathology. The headache of fever and toxæmia is generalised or mainly frontal. Severe frontal headache with rise of temperature usually indicates the onset of a specific fever. Such headache disappears if the child becomes delirious, whereas it persists if due to organic disease. The headache of meningitis is a fair measure of the severity of the disease. It bears no proportion to the amount of fever, is more constant than intense, and does not vary with position. Acute headache with an infrequent, irregular pulse is diagnostic. Acute headache without vomiting excludes it. But headache, intolerance of light, vomiting and constipation do not always indicate tuberculous meningitis. Roughly the causation, based on localisation, is as follows:—

*Frontal.*—Anæmia, toxæmia, eye-strain, astigmatism, naso-pharyngeal affections, over-growth.

*Vertical.*—Anæmia, hysteria, epilepsy, meningitis.

*Parietal.*—Carious teeth, ear troubles.

*Occipital.*—Epilepsy, meningitis, eye-strain, pharyngitis, cervical caries, otitis media, decayed molars, cerebellar disease.

*Supra-orbital or frontal.*—Disease of the nose or frontal sinus.

A localised headache is suggestive of tumour or abscess. It is sometimes relieved by the remedies useful in simple headache, for the pain is due to hyperæmia or irritation. Possibly the pain indicates involvement of the meninges. In inflammation of the tympanum, middle ear or Eustachian tube the pain is referred to the external ear and its neighbourhood. It is more a “dolor capitis” than true headache. Mental overstrain is often associated with phosphaturia.

*Toxæmic, bilious or gastric headache* generally begins in the morning, perhaps because of the comparative inactivity of the kidneys and



respiratory system during sleep. The child is languid, has no appetite for breakfast, and exhibits a coated tongue and indicanuria.

*Morning headache*, frontal in distribution, may be due to rapid growth. It is most common in rheumatic and neurotic boys at 9-12 years of age. It is aggravated by work, decreased by rest. The headache of anæmia and debility is relieved by recumbency and increased by the upright posture. Giddiness and nausea are produced by getting up suddenly.

*School headache* is due to vitiated atmosphere, mental overstrain, and errors of refraction. Deficiency of animal proteins and excess of carbohydrates in the diet are predisposing causes. If due to fatigue or overwork it comes on in the latter part of the day. The majority of children do not work unduly hard at school, so mental strain is not a frequent cause of headache and breaking down, except at examination periods.

**Migraine**, a corruption of hemicrania, occurs in periodical or quasi-periodical attacks, usually early in the day. Hereditary transmission or a family history of headaches, neuralgias, gout and neurotic affections, is often present.

H. Neumann gives the age of onset of 43 cases as 9 in the first, 21 in the second, and 13 in the third 5 years of life.

The *prodromata* are cloudiness of vision, hemianopsia, photopsia, teichopsia, and central or lateral scotoma. Sometimes a star is seen near the fixation point, zigzags, catherine wheels, or a fortification spectrum, which may be coloured. Occasionally an aura, like an epileptic aura, spreads up the arm. It is much more prolonged and deliberate in its ascent, and is followed by hemicrania, and perhaps aphasia for some hours, much more severe than in minor epilepsy. Prodromata last for a few minutes to half an hour, subsiding as the pain comes on. Pain may be the first sign. It is unilateral, increases in severity for some hours, and may be intense. There is no unconsciousness; no shrieking, as in meningitis; and it subsides gradually, the patient then falling asleep. Nausea is common, vomiting infrequent. Frequently the symptoms consist of fever, drowsiness, moderate headache and vomiting. They may simulate meningitis. The pain may be so severe as to cause delirium and stupor, and suggest post-epileptic mania. A boy, aged 12, was subject to attacks for a year. The pain was in the right frontal region, sufficiently severe to make him cry out and light-headed, and associated with visual hallucinations. He came under treatment for chorea.

The *symptoms* are suggestive of primary vasomotor constriction of the posterior cerebral artery or its occipital branch, followed by dilatation. The pain can be relieved by compression of the carotid. During the stage of vaso-constriction there is no headache. Occasionally migraine and epilepsy alternate in the same patient, and migraine may cease when epilepsy develops. This is a rare event. Possibly the connection depends



on the susceptibility of the brain in childhood to irritation and convulsions. The pain of migraine may be of such severity as to cause convulsions, which may eventually result in epilepsy, in spite of the gradual subsidence of the attacks of migraine. As a rule epileptics are remarkably exempt from headaches.

*Paroxysmal recurrent headache*, frontal or occipital, is a sub-variety of migraine although it is not unilateral. They are both recurrent, due to similar causes, separated by intervals of poor health, and may alternate in the same child. In gouty cases the child may be quite well between the attacks. Severe neuralgic headaches occur in dyspeptic children during the second dentition. They produce intolerance of light, nausea and vomiting. In girls they may recur at puberty and at each menstrual period, with some gastric disturbance but little uterine pain.

*Ophthalmic Migraine*.—*Syn.*: *Periodic Oculo-motor Palsy*—*Transient Ophthalmoplegia Externa*—*Recurrent Palsy of the Third Nerve*—*Fleeting or Recurrent Palsy of Ocular Muscles*.—This begins with periodical attacks of hemicrania, nausea and vomiting; followed by oculo-motor palsy which at first disappears between the attacks and later on becomes permanent. It may occur in infancy and old age, but is most common in young adults. Autopsies have shown plastic exudation or tuberculous granulations round the trunk of the third nerve, and fibrochondroma or fibroma of the nerve or nerve sheath.

*Treatment of Headache*.—Procure elimination in toxæmic cases by calomel or grey powder; and give a mixture of rhubarb and soda, if there is any digestive trouble. Attend to any local cause. Do not overlook albuminuria and chronic interstitial nephritis. For migraine prescribe complete rest and quiet in a warm, darkened room. Inducing vomiting may cut short an attack. Cooling lotions to the scalp and a mustard leaf, or even a leech, over the mastoid, may do good; or hot fomentations or an ice bag. Lower general blood pressure and intracranial pressure. Lumbar puncture might be tried if all other measures failed to give relief. It is often used in cases of organic cerebral disease. Trinitrin is useful, with strychnia, in anæmic headaches and migraine; amyl nitrite, if vasomotor spasm is indicated by pallor; ergot and strychnia, if there is general vaso-dilatation. Chloroform inhalations, valerian and camphor, sal volatile, ether and opiates are beneficial. Iron and tonics are required for the headache of debility and overgrowth; aspirin and sod. bromid. for rheumatic neurotics; and in all cases regulation of the diet and mode of life in accordance with the causation of the headaches.

**Sleep**.—Sleep is a regularly recurring function, not merely cerebral. Goltz removed the cerebrum of a dog. It survived  $1\frac{1}{2}$  years and showed regular alternations of sleep. Modifications depend on exhaustion, toxic substances such as morphia, cold as in the hibernation of animals and



plants, the interruption of nerve conducting paths in cerebral tumour, and the abolition of sensory stimuli in cutaneous anæsthesia.

Many growing children suffer from too little sleep, none from too much. A child needs more than an adult for its vital processes are much more active while awake, and a longer interval is necessary for recuperation as well as for mechanical rest of growing structures. At puberty and in children of active intelligence sleep is most essential. The stress and strain of modern life must be prepared for by training in habits of sleep, by ensuring proper development of the body and brain, and by guarding against the effects of strain in school life.

The newborn babe falls asleep after its bath and only wakes in response to sensations of hunger, thirst, cold, wet or colic. Its wants attended to, it falls asleep again. If labour has been much prolonged sleep may approach semi-coma for 1-2 days. Actual cerebral injury produces fits or rigidity. During the first 24 hours the babe is almost constantly asleep. In the second 3 months the intelligence develops and the child sleeps less. By the fourth month it may remain awake for a few hours at a time without injury, but should still sleep for 18 out of 24 hours. In the second year the child should sleep 12-14 hours at night and about 2 hours in the middle of the day. By the fifth year the day sleep may be omitted, if the child has the same hours of sleep at night.

The normal attitude is on the side, curled up in the intra-uterine position, with the eyes and mouth closed. The babe should be trained in habits of sleep from the time of birth. Quiet, darkness, warmth, dryness and suitable diet are needed. One period of sleep at night should last 5-6 hours. After the sixth month this should be prolonged to 8-9 hours, the child only being fed 6 times in 24 hours. Allow no sleeping at the breast or bottle, no rubber teat, and no nursing or rocking. Do not encourage profound silence in the room or house, nor sleeping out of doors at the expense of indoor rest. Do not let ventilation be sacrificed to darkness. Do not encourage special habits for the production of sleep, e.g. sucking the thumb or a corner of the sheet, covering up the face, the sitting posture or lying on the stomach. Wake the child to pass water. It will drop off to sleep again at once. After the first 2 years of life sleep is much more profound.

*Excessive sleepiness* is occasionally a sign of imbecility or due to drugs. Paregoric and soothing syrups produce constipation. White bryony, sold in the form of white globules, causes diarrhœa. Drowsiness is a sign of the onset of measles and other illnesses, and may be extreme in cerebral tumour. Some neurotic children are difficult to wake up in the morning and are not mentally active for several hours.

*Wakefulness* is abnormal. In infancy it is a disturbed and restless sleep rather than true insomnia. It is due to colic, excess or insufficiency of food, night feeding and bad training. Some children lie awake for hours and then sleep normally. At all ages it may be caused by painful or



irritating affections, e.g. teething, toothache, skin irritation, ear troubles, cough, abdominal complaints, adenoids and enlarged tonsils, etc. Sometimes it is due to too little or too much clothing, cold feet, heat, insufficient ventilation, palpitations, febrile attacks, or the starting pains of hip disease. Neurotic children sleep badly, wake at the least noise, and are soothed with difficulty. Restlessness and insomnia are common in rickets, tetany, laryngospasm, head banging, anæmia and dyspepsia. Overstimulation, nervous excitement and over-fatigue are common causes. Quite half the cases are of digestive origin.

Treat the cause, if discoverable. In other cases rely on wholesome regular diet, hygiene and exercise, and training. Put the child to bed at a regular time, in a dark, quiet room. The nurse or mother may sit, holding the child's hand, and sing monotonous tunes in a low voice. If the child is very nervous or suffers from pavor, allow a night light and let the attendant sleep in the same room or an adjacent one with the door open. A warm bath should be given at bedtime. Give no food during the night and omit supper, if there is any pavor or digestive trouble. Bromides are needed for pavor, chloral for cough and laryngospasm, and opiates for actual pain; chloral, bromides, codeia or trional for acute febrile disturbance.

*Sleep at School.*—Usually the hours of sleep are too limited but the evil effects are counter-balanced by long holidays. Yet many children suffer in later life from over-cerebral activity during school life and this is undoubtedly due to insufficient sleep. It is remarkable what a number of children, regarded as exceptionally brilliant at school, prove failures as adults. The early rising and morning chapel, followed by a repetition lesson before breakfast, are by no means advantageous to mental and physical development. Individual requirements of sleep vary.

Boarding-school life should begin at 8-10 years of age, even a year earlier in some cases, and from then up to the twelfth year the child should go to bed at 8.30-9.0 p.m., and get up at 7.30 a.m. in summer, and at 8.0 a.m. in winter. Allow  $10\frac{1}{2}$  hours sleep at 11 years of age and reduce the amount by a  $\frac{1}{4}$  hour yearly, or  $\frac{1}{2}$  hour every 2 years, up to 19 years of age. From 13-16 years of age the child should go to bed at 9.0 p.m. and get up at 7.0 a.m. in summer and  $\frac{1}{2}$  hour later in winter. After that the retiring hour may be 10 p.m. It is absurd to suppose that habits of self-indulgence are induced by long hours of sleep. Nature is a reliable guide during the early years of life. After puberty is safely over, more spartan habits may be encouraged. The distinction between junior and senior boys must be based on age and not on mental activity. A clever, forward boy requires as much sleep if not more than a backward one of the same age. The sleeping apartment must be darkened, so that the child is not awakened by light in summer, and kept quiet by suitable discipline. Boys who retire at a later hour should not occupy the same apartments as those who are already in bed. Insufficient sleep produces a tired aspect, mental inefficiency, retarded



growth and development, sleepiness and inattention at lessons, nightmare, somniloquence, pavor and somnambulism. It must not be forgotten that growth takes place while in bed. Later results are the effects of cumulation and may not be evident for months or years. They are indicated by neurasthenia, brain fatigue and exhaustion, inability to stand strain, headaches, insomnia, neuroses, and possibly chorea and epilepsy.

**Nightmare.**—Children sometimes scream out in their sleep and frequently wake up at night frightened, excited or terrified. Dreams may be pleasant or unpleasant. Nightmare, somnambulism, teeth grinding, pavor nocturnus and pavor diurnus are all more or less allied. There is no strict dividing line and the same child may suffer from all. The chief etiological factors are a neurotic heredity and predisposition, ill-health, febrile states, the various causes of respiratory obstruction leading to deficient oxygenation of the blood, poisons such as lead, belladonna, and alcohol at bedtime, and gastro-intestinal disturbance.

Nightmare is a dream in which there is great distress in consequence of a feeling of inability to move and save oneself from a horrible or fatal situation. The dream is unpleasant and terrifying, and the child wakes up frightened and excited. The mind is clear, the attendants are recognised, and the details of the dream are often remembered next day. Nightmare may occur at any age. There may be several attacks during the same night, in the same or a different form. In recurrent attacks on subsequent nights the same dream may be repeated. Asphyxial and gastric causes are the chief factors in its production. Many attacks are due to a late meal, over-feeding at night, ill-digested food or constipation; others depend on adenoids, large tonsils or nasal obstruction; and possibly a few are due to reflex irritation. The type of dream is based on some visual or auditory impression during the waking hours.

**Somnambulism** is a type of nightmare of similar causation. The term includes all varieties of motor action during sleep and is not limited to "walking." In this state the child can do many things impossible to him while awake, such as climbing and walking along dangerous parapets. Or he will get out of bed, look for things under the bed, empty or rummage in drawers, open windows, walk downstairs, and carry out almost any action, even dangerous ones to other people, including getting a knife and stabbing another child. The eyes are wide-open, the movements deliberate and purposive, but consciousness is in abeyance and attendants are ignored completely. A bright light or sharp order will send the subject back to bed. Visual hallucinations are exceptional. A 7-year old boy saw "things crawling on the wall." Next day there is no remembrance, or only a very faint one, of the occurrence. Late suppers and school work are the common exciting causes. These children should be fastened in bed with a belt round the waist and a ring at the back. Pass a bandage through the ring and tie it under the bed.



*Somniloquence*, or “*talking in sleep*,” can be regarded as a mild form of somnambulism. Children, on first going to school, constantly talk over their lessons, especially sums, in their sleep. In a severe form long conversations are held with imaginary people and the condition is analogous to pavor.

*Teeth grinding* is similar in character. It is apt to occur in infants during illness, and in neurotic and imbecile children. Occasionally it is associated with other motor habits, e.g. the child “scratches his stomach,” “worries at himself,” “turns over and kicks about.” In this respect it is allied to head burying and similar complaints. All the teeth may become bevelled down to the same level.

**Pavor Nocturnus or Night Terror.**—An excellent description of this peculiarity is given in Lamb’s Essay on “Witches and other Night Fears.” It is a type of sleep disturbance characterised by emotional distress or a nightmare exaggerated by the neurotic disposition of the child. Much attention has been devoted by Guthrie and Coutts to a differentiation between nightmare and pavor. Certainly between the two extremes the difference is considerable, but there are so many intervening degrees of the same states that on the whole it seems advisable to regard them as similar in their causation and mode of production, though perhaps of different prognostic import. In one sense it is a paroxysmal neurosis of the higher centres. Emotional distress, hallucinations, abeyance of consciousness and lack of remembrance next day are the chief features.

The attack comes on in the early part of the night, an hour or two after going to sleep. The child suddenly starts up with a piercing shriek and is found sitting up in bed, standing on the floor or hiding in some part of the room, in a state of terror and mental confusion, with sometimes a sense of suffocation. Sometimes he is very pale. He may scream with fright, or cry out inarticulately and unintelligibly. He is oblivious of his surroundings, does not recognise his nurse though he may cling convulsively to her, and has hallucinations of vision, rarely of hearing. His pupils are dilated, eyes wide-open and staring, the body trembling, hands clenched or moved as if in self-defence, and he sweats freely. A vivid description was given by the mother of a 5-year old boy :—“He started up in bed with a piercing shriek and shouted out, ‘The devil is here ; take the knife away for he has cut her head off.’ His eyes were like saucers, his body trembled and he was all of a sweat.”

The terror is usually undefined and does not vary in character. Often it takes definite shape and the child cries out, “Look, look !” “They are coming !” or sees “boys and horses about the room,” “worms crawling on the pillow,” “ugly faces with masks on,” etc., due to visual hallucinations. Sensory disturbance was present in one child who complained that “dicky birds were biting her hands and feet.” Vertigo is not infrequent and is probably auditory in origin.



The attack lasts a few minutes to an hour. Frequently the child falls asleep without return of consciousness. If he becomes conscious, he may remain frightened and under the delusion, or talk of the hallucination and later ask to be put back to bed. Profuse micturition or defæcation may occur at the end of, or even during, an attack. Usually there is no remembrance of the attack next day; sometimes a faint recollection or consciousness of something horrible or unpleasant. The child shirks talking about it but may describe the nature of the hallucination, if his confidence is gained. Except in febrile states there is rarely more than one attack a night. It may recur every night, at varying intervals or quite irregularly. The hallucination is almost always the same.

Pavor has been divided into (1) Asphyxial and (2) Primary Cerebral types. The *asphyxial type* is allied to nightmare and the terror is subjective. The attack develops slowly, comes on an hour or more after falling asleep, and is characterised by a feeling of suffocation, mental confusion, hallucinations, amnesia and muscular weakness in consequence of the asphyxia.

The muscular debility is shown by the inability to articulate clearly and the clumsy movements. The incidence and severity of these attacks depend on the degree of respiratory obstruction. Fright and hallucinations may occur in any disease leading to deficient oxygenation. On the other hand, in many instances the cause appears to be gastric or intestinal. In such cases we must attribute the dyspnœa and sense of suffocation to reflex stimulation of the vagus, if we accept the asphyxial view of their pathology.

The *primary cerebral* or *idiopathic type* is the one in which no asphyxial or alimentary cause is discoverable, and a definite predisposing factor is present in the shape of a neurotic or alcoholic parentage, a rheumatic diathesis, or past infantile convulsions. These children are neurasthenic; nervous, irritable, impressionable, excitable and often anæmic. They sleep lightly, restlessly, and wake easily. The terror is objective and due to over-excitement of the cerebral cortex in a nervous child. The exciting cause is the same as in other varieties of disordered sleep, viz., school work, pantomimes, dark rooms, variable shadows, gruesome pictures or tales of ghosts. In normal children ghost tales and horrors do not induce attacks; possibly they merely formulate the vision in the susceptible child. Occasionally the attacks are due to fever, tuberculous meningitis, or head injury.

*Etiology.*—Pavor is most common in the fourth year of life, the age when adenoids are likely to produce the greatest amount of respiratory obstruction; fairly frequent up to beginning of the seventh, coincident with the commencement of school life; less common up to the twelfth year and rare subsequently. In half the cases under my care a digestive cause has been present. The digestive disturbances, consequent on carious teeth, the second dentition and late meals, and school life are often



combined. It is frequently found that more than one factor is present. The sex-incidence is 3 females to 2 males.

*Complications.*—As already stated all forms of sleep disturbance may occur in the same child at different times. In the cerebral type of pavor we are likely to find the usual signs of neurasthenia, especially headache, palpitations and muscular fatigue.

Abnormally bad temper, habit spasm, enuresis, moral insanity, fits, hysteria, migraine and insanity may be present ; but the pavor should then be regarded as a symptom of these affections and not these looked upon as complications of pavor.

*Diagnosis.*—Never accept a case of pavor as primarily cerebral until all other causes have been excluded. Do not confuse nightmare with pavor. Coutts draws a striking distinction, unfortunately not always confirmed in practice, but frequently of great use. He holds that in pavor the child sees visions, in nightmare he dreams dreams. Nightmare may occur in any child, at any age, in several attacks during the same night, and without grave terror. It is associated with ill-health, with recollection of the nature of the dream, and with recognition of the nurse and surroundings. Night terror occurs in the neurotic or in children with a family history of neuroses, is rare after 12 years of age, is independent of ill-health, and usually in single attacks. There is lack of consciousness of surroundings, grave terror and emotional distress, and no recollection of the attack next day. The nature of the vision is constant, while the dream in nightmare may vary and take several forms in the same night. Pavor is distinguished from epilepsy by the absence of a fit, tongue biting, and subsequent stupor or intellectual torpor.

*Prognosis.*—Sleep disorders are commonly symptomatic and the outlook good. Somnambulism may persist in those of neurotic ancestry and lead to fatal accident. The predisposing causes of cerebral pavor may produce tics, migraine, neurasthenia, hysteria, epilepsy and insanity in later life. Imbecility or severe brain disease is rarely present. Pavor does not predispose to epilepsy.

*Treatment.*—Give an aperient, attend to the diet, and regulate the mode of life. Ventilate the sleeping apartment thoroughly, allow the use of a night light, and let the nurse sleep in an adjacent room with the intervening door open. Treat local causes of asphyxia and irritation. Permit no physical or mental strain, limiting school work to the morning hours ; no late meals and no parties.

The child should sleep on a hard mattress, with no heavy bed-clothes, and have a bobbin fixed to the spine to keep it off the back. Neurotic children must be protected from unpleasant sights and shocks, ugly pictures, gruesome tales, threats, excitement, and some of the appalling toys which are wrongly considered suitable for children. Attend to the digestion, giving a mixture of rhubarb and soda, and keep the bowels open. Give a



dose of bromide, bromide and chloral, or phenazone at bedtime; or bromides or phenazone grs. 1-4 t.d.s. Iron is needed if there is anæmia.

**Pavor Diurnus.**—This is a day-terror of cerebral origin, which may be associated with or replace pavor nocturnus. In some cases there is definite irritation, e.g. constipation, worms or colitis. There is a sudden, unaccountable attack of screaming and terror. The child rushes to someone for protection and cannot be pacified. The attack may come on in the midst of play, or while the child is apparently awake though possibly in a dreamy state, and with visual or auditory hallucinations. There may be dread of the mother or nurse, and even temporary mania. In milder attacks there is sudden pallor, sometimes a vacant look, but no loss of consciousness. They last from a few seconds to 15 minutes. Attacks may occur several times daily, or only at intervals of a week or two. In young infants they may take the form of *paroxysmal screaming* and be put down to bad temper. An anæmic dyspeptic boy, 16 months old, had frequent screaming attacks for 3 weeks. They were of sudden onset and cessation, and followed by much trembling for 10-15 minutes. They ceased as soon as the dyspeptic state was cured.

Day terrors, day somnambulism and narcolepsy may be associated. The outlook is less favourable than in other forms of pavor for they appear to indicate a high degree of the neurotic disposition, and a greater liability to neurosis in later life.



## CHAPTER LVIII.

### DISORDERS OF THE HIGHER FACULTIES.

*Neurotic Children—Precocity—Neurasthenia—Psychasthenia—Hysteria—Mental Deficiency—Mongolism—Insanity—Speech Defects—Deaf-Mutism—Word-Deafness—Word-Blindness—Idioglossia—Stammering.*

**Neurotic Children.**—The neurotic disposition is inherited. It can be modified by training and environment but not wholly eliminated. This peculiarity of the nervous system, rather than the normal instability of the nerve centres in early life, is the primary cause of bad habits and neuroses. The chief predisposing factors are those common to all children; viz. defective control by the higher over the lower centres, and imperfect definition of the motor tracts leading to inco-ordination and spasmodic affections. The effects on the nervous system of malnutrition, rickets, alimentary disorders, rheumatism, various toxæmias and reflex irritation, are partly predisposing and partly exciting causes.

These children are subject to general impairment of metabolism and vitality. Emotional excitability is abnormally keen, easily stimulated and controlled with difficulty. Whether restrained or unrestrained, it is physically and mentally exhausting. It may develop into a definite neurosis if the will power is also defective. Guthrie recognises a *restrained* and an *unrestrained emotional type*. In the former the emotions are as strongly controlled as they are felt. Though intelligent and observant these children are so reticent that they are thought dull and stupid. They are sensitive, shy and proud; desirous of sympathy and affection; possess no sense of humour; are brooding, gloomy, solitary and revengeful; and develop abnormal scruples and phobias. Children of the *unrestrained emotional type* are normally or unduly intelligent, timid, high spirited, full of restless energy and easily discouraged. They are impetuous, enthusiastic but wanting in judgment, extraordinarily vain, with a saving sense of humour, and do not always take themselves seriously. Thus, in one variety there is emotional stress with expression thereof, and in the other stern repression of the signs of excessive emotion. Under suitable conditions hysteria develops in the unrestrained and neurasthenia in the restrained type.

*Symptoms in Babies.*—Hypersensitiveness of the special senses is the chief sign of a neurotic disposition. The baby may refuse substitutes for the



breast, rebel against slight changes in the diet which may be imperceptible to the adult, express repugnance to certain flavours and disgust at the presence of the least particle of solid or gritty matter. This objection to solid food may be so great as to cause nausea, "gagging," gargling and vomiting, and prolonged bottle-feeding. Occasionally it depends on inco-ordination of the muscles of deglutition. It may lead to idiosyncrasies in food and drink, and the desire for unsuitable foods. Hot greasy fat and cod-liver oil are intensely disliked by neurotics of the unrestrained type; and the necessary amount of fat must be given in the form of butter, margarine, cream, dripping, or olive oil in salads. Pica (p. 250) is not uncommon in the first year of life.

Hypersensitiveness to light is indicated by the unduly early acquirement of normal perception and alarm at the sight of unaccustomed objects and people. Co-ordination of the ocular muscles is established in the third month, but a precocious child may follow a bright light and turn its head as early as the sixth day. People and objects are recognised in the sixth month. Occasionally there is abnormal sensitiveness to colour, and the expression of alarm at red or, less often, yellow.

In the first 24 hours the child is apparently deaf. In 2 weeks it appreciates loud sounds, and in two months it recognises the direction of conveyance of sound and turns toward it. Acuity of hearing develops more quickly in some infants and they may awake at the slightest sound. Neurotic babies are very sensitive to loud or discordant noises, and often frightened thereby. Such sensitiveness may persist and be a cause of the timidity, tearfulness and unsociability of some children at noisy parties. It may depend on the musical sense and be stimulated only by unmusical sounds. Loud noises often terrify children. The sense of smell may be abnormal and certain odours may produce paroxysmal asthma, syncope, nausea or vomiting. Cutaneous hypersensitiveness is shown in the tendency to erythematous and urticarial rashes. The irritation of tight bands or unsuitable garments may cause insomnia, habit spasm and other disorders.

Other signs, more obvious in *older children*, are fretting, home-sickness, timidity, fear, morbid apprehensions, worry, and perversions of religious and moral emotions. Day-dreaming is an exaggeration of the imaginative faculty to such an extent that the dream is mistaken for reality. It may lead to romancing or lying. Usually the dream relates to the child's own personality. *Timidity* is not necessarily cowardice. It may depend on imagination, the fantastic shapes evolved from lights and shadows; on fear of reproach, harsh words, anger or ridicule, or the disgrace of corporal punishment; or fear of the unknown or of impending trouble. Ridicule will frequently prevent a child attempting even simple feats or ordinary book tasks. In its severest form the child is afraid of ordinary animals, sights and sounds. These children must be encouraged. Their confidence must be acquired, night lights allowed, and their fears relieved by common



sense explanations of the phenomena causing them. Day-dreamers must be kept occupied with work or play.

**Mental or Intellectual Precocity** is unduly rapid development, relative to an artificial standard applicable to children of the same race, family, sex, age and environment. Like natural ability it follows the laws of variation from the mean and is not necessarily abnormal or pathological. The variation from the mean may take place in other directions and result in imbecility, criminality, sexual perversions or moral insanity, all of which may occur in the same family as that of the genius.

*True precocity* is a variation from the normal, dependent on an inborn or hereditary tendency and modified by environment. It must be distinguished from false or *artificial precocity*, due to external influences, over-pressure, town life, and excessive stimulation of the mental faculties. External influences modify but do not cause true precocity.

Precocious children are usually the first or last of large families, or born of parents of greatly dissimilar ages. In these respects precocity is like Mongolism. Only children show precocity because of their surroundings, being constantly in the society of their elders and over-stimulated. Overgrown children are liable to be precocious, through being dressed in accordance with their size and associating with older companions. Mental troubles and neuroses are frequent during the period of rapid growth of the brain, from 1-7 years, and at puberty, and are rare in the intervening period. Care should be taken not to over-stimulate the brain at these periods.

Precocity may be of good or bad omen. It does not imply prematurity and early decline of the intellectual powers, nor is it incompatible with long life and prolonged mental ability. The idea that precocious children die young, or become insane because of their precocity, is not borne out by facts, at least as regards the truly precocious ones. But excessive ability of an acquired type is often associated with defective heredity and may end in dementia præcox, neurasthenia or hysteria. Extraordinarily clever men have generally surpassed ordinary children in their early years. This is notably the case among artists and musicians, while mathematicians and philosophers are of later development. In the course of intellectual growth sense impressions, mental images and abstract ideas develop successively. Genius does not always specialise and in its highest form (*intellectus universalis*) includes everything, except perhaps music and art. As a rule the great men of the world are drawn from the precocious children, but fond mothers can console themselves by the fact that genius is not always recognised in early life and the supposed dullard is sometimes a boy of remarkable originality. Many great men are not precocious in childhood. Some children develop much later than others. Yet, as a rule, the greater the ability in early life the greater is the prospect of distinction in later life, provided that failure does not ensue from lack of opportunity, perseverance,



ambition or other moral qualities, and that the precocious brain is not spoilt by overpressure or uncongenial work.

Mentally diseased children may be precocious and precocity may be associated with delicacy, though by no means frequently. Constitutional weakness is not uncommon in the "Wunderkinder" at one end and in imbeciles at the other end of the scale. Imbeciles are liable to tuberculosis. Delicacy may induce artificial precocity, through compelling the child to a less active life, with more constant stimulation of the brain by reading and adult companionship. It is generally found in schools that the taller children of the same age are in the higher classes and that dullards are below the mean height. "Wunderkinder" are exceptional, e.g. calculating prodigies and those with extraordinary memory, musical capacity, excessive manual skill or muscular power, mimetic faculties, etc. Some of these have normal or increased general mental power, while others (idiot savants) are mentally deficient and usually die young. A minor degree of brilliance in one direction may be associated with dulness in others. Precocity and natural ability follow the same laws of health and physical development. Artificial precocity is the most likely to be worked out and end badly.

*Treatment.*—True precocity may be encouraged in its natural bent and should not be thwarted, as long as there is no excessive stimulation. Artificial precocity must be repressed and the child's energies directed into other channels such as an open-air life, games, botany and the study of animal life. A liberal allowance of sleep is essential. Erratic precocity is the most dangerous and requires assiduous care. Aim at complete all round development and map out the daily routine of life. Guard against emotion and excitement. Inculcate physical, mental and moral discipline. Each case should be treated on its merits, and delay in mental development must not be considered a drawback.

**Neurasthenia.**—Neurasthenia, psychasthenia and hysteria can be differentiated from each other, though they are often combined and present many intermediate forms. They may occur in early life, especially about puberty and in early adolescence, but are more frequent at later ages. The predisposing cause is a neurotic heredity, a capacity for feeling emotions, sensations and physical stimuli to an abnormal degree and on inadequate provocation in comparison with a normal child. The exciting causes are physical and psychical depression such as result from malnutrition, the toxins of influenza and other infections, fear, shocks, injury, lack of sleep, irregular mode of life, over-stimulation of the brain and imagination, family jars and quarrels, and undue strain on the emotions.

*Neurasthenia* is a state of irritable feebleness with general depression of the cerebrospinal functions; a combination of irritability and exhaustibility of the higher cerebral and spinal nerve centres, or a hypersensitive-ness of the higher centres and ready exhaustion of physical and nervous energy. Affected infants are abnormally easily frightened, startled by noise



or bright light and utter reflex cries, struggle or cry violently in the bath, are constantly wailing and sleep lightly. In young children fear of strangers, animals and new toys is the most marked symptom.

The chief symptoms are somatic and psychical. Nutrition is impaired and the general health indifferent or bad. The main objective and permanent signs are exaggerated deep reflexes, normal or deficient corneal and pharyngeal reflexes, variable pupils, tremor of the eyelids and incomplete closure on shutting the eyes, tremulous hands, cold hands and feet, hurried or stammering speech, variability in the pulse rate, vasomotor troubles and fleeting erythema. Downheartedness, disinclination to play or work, restlessness, rapid fatigue, and undue emotion are commonly present. Other symptoms are subjective and often paroxysmal in character, such as headache, syncope on fright or fatigue, vomiting on waking or going to school, anorexia, constipation, enuresis, frequent micturition and stammering of the bladder, palpitations and arrhythmia; convulsive and alarming cough with cyanosis, choking and vomiting, simulating pertussis; exhibitions of wild rage, convulsive sobbing or laughter; egotism and self-analysis. Dreamy states take the place of hysterical fits.

*Attacks of convulsive rage* are most frequent at 1-5 years of age. The child is unable to cry because of arrested expiration, due to spasm of the expiratory muscles. The face turns pale, body rigid, eyes upward, and the child falls backward unconscious. At the onset she may strike out wildly with the fists, roll on the floor and kick, or throw herself down and scream violently in the public street. Sometimes the face is turgid and bluish, the whole body trembles, and the child suddenly ceases to scream and becomes rigid, with the mouth wide-open, and comatose. Then the spasm relaxes and a loud cry is uttered. There is rarely involuntary micturition and defæcation. It differs from the expiratory apnœa of laryngospasm in the absence of inspiratory stridor and of galvanic over-excitability. The attack can often be stopped by punishment or threat thereof at the onset, that is as soon as the child attempts to hold the breath, and can be shortened by dashing cold water in the face, a slight smack or cutaneous irritation.

Neurasthenia is acquired and curable. It is usually gradual in onset and chronic in course. It may be temporary, and cured by removal of the cause. Sometimes it is localised in one special symptom and difficult of diagnosis, or it may be an early stage of organic nervous disease. The prognosis depends upon the heredity and environment.

**Psychasthenia** is a variety of neurasthenia in which the mental symptoms predominate. It may be associated with unusual intellectual gifts, defective intelligence, or lack of moral sense. It chiefly affects timid, impressionable children; and may be regarded as the climax of hereditary predisposition, having its basis in an inherited and more or less degenerate nervous constitution. Hence it can never be regarded as completely curable. Usually it comes on at puberty or shortly after, but may begin as early as the



eighth year. Half the cases are under 20 years old. It is more common in females and there is often no apparent exciting cause.

The child develops morbid scruples, increasing mental uncertainty and hesitation, lack of initiative power or acting promptly, auto-analysis, and excessive emotional reactions. Next it commences imperious strange acts such as frequent washing of the hands, aimless rummaging, and verification of unimportant matters (tics) ; or stays in a state of rumination or reverie.

Finally, it develops fixed ideas, persistent obsessions and phobias. Uncontrollable actions depend on these uncontrollable ideas, though they are realised by the patient as morbid. The will is enfeebled and irresolute, the child being incapable of fixing either attention or energy. In manner there is eccentricity and in action clumsiness. In spirit the joyousness of youth is replaced by melancholy or hypochondriasis.

The affection may take several years to develop fully, and often there are periods of remission. The child may know when an attack is coming on. She becomes flushed, restless, unable to keep still, jerky and agitated ; or occasionally dull, inert, listless and mumbling.

The obsession or phobia is well marked and assumes various types in different cases. It may be persistent or vary in different attacks. The chief types are :—(1) Doubt, hunting out objects, counting and naming ; (2) Metaphysical mania or exaggerated scruples of cleanliness and exactness, giving rise to uncontrollable desire to wash and even self-mutilation ; (3) Agoraphobia, claustrophobia, dread of travelling, an intense desire to roam, truancy and running away from school ; (4) Fear of light, fire, sharp objects, clothing, dirt, etc. ; (5) Criminal desires, perhaps theft ; (6) Sexual perversity ; (7) The alcohol and drug habit ; (8) Self-accusation. The disease is progressive, unless treated (p. 780), and liable to end in suicide during depression, hypochondriasis, melancholia, or delusional insanity.

**Hysteria.**—In this we find a lack of will power, combined with a process of self-suggestion and hypnosis in the production of a great variety of symptoms, which are sometimes extraordinarily difficult of differentiation from organic disease. It is a mental disease of the “unconscious or sub-conscious mind,” an aberration of the cells governing function and nutrition. It is curable by and produced by suggestion, and must be distinguished from imposture, simulation, malingering and organic disease. A normal child does not simulate disease. Accepting this view we must consider some of the symptoms, generally regarded as hysterical, as signs of added neurasthenia. True hysterical symptoms depend on intellectual or emotional psychic causes. The hysterical child is egotistical, desires importance and attention or admiration, is emotional, and has a lively imagination. Explosiveness and excitability of the higher nerve centres are combined with lack of control over emotional display and subsequent exhaustion. It is a cerebral, not a cerebrospinal affection. The symptoms



may come and go suddenly, subside rapidly and completely, be cured suddenly after a duration of years, are dominated by mental and moral influences, and are compatible with perfect physical health and nutrition.

*Etiology.*—The predisposing and exciting causes are the same as in neurasthenia. Apart from the latent condition the exciting cause is usually quite inadequate. Susceptibility is greatly influenced by training and environment, by undue fostering of the emotional side and by over-indulgence. Association with neuropathic and capricious parents, and imitation, are important causes. It is as common among the poor as the rich, in isolated rural centres as in towns, and at any period of the year. It usually begins at 8-12 years of age, rarely before 5 years, but may occur as early as 3 years. Most of the cases in the very young are more truly neurasthenia. It is more common in girls. Boys are more affected in childhood than in later life and more severely than girls. It is more difficult to recognise in boys, for organic disease is better simulated and boys play the game more thoroughly, being afraid of being found out or laughed at for girlish tricks.

*Symptoms.*—The onset is sudden, with full development of symptoms which are usually limited to one and that exaggerated in type; generally motor, rarely sensory. Many symptoms arise from suggestion or imitation; or are due to auto-suggestion, persisting after the original cause has disappeared. Certain general symptoms may be present as well as the particular one that attracts attention, but hysterical stigmata are rare in the very young for the child's mind is simple, and less common in older children than in adults. At puberty they approach the adult type.

The *sensory* disturbances include anæsthetic spots, anæsthesia of the pharynx, conjunctiva and skin, hemi-anæsthesia, or a sleeve-like distribution on one limb; hyper-æsthetic areas not corresponding with any special nerve or nerves, and hysterogenetic zones or tender points; concentric limitation of the field of vision; headache, clonus and localised pain; and globus hystericus. Deafness, deaf-mutism, mutism and blindness are rare. Sometimes there is aphonia or dysphonia, which is absent on coughing and singing. The speech is whispering for the cords are imperfectly approximated. Dysphagia may be present in neurotic infants about the time of weaning and due to objectionable food, hurried feeding or nervousness. In older children there may be vigorous but unavailing attempts to swallow.

The *psychical* symptoms take the form of putting out the tongue and spitting, attacks of shouting and screaming, exhibitions of anger and crying without sufficient cause, apparent fright, in which the hands are clenched, uncontrollable laughter or giggling, mimicry of animals, stuporous states, and sexual or erotic troubles.

The *motor* symptoms include convulsions, spasmodic seizures and contractures, paralyses and tremors. Some relapses in chorea are hysterical.



In hysterical convulsions there is no self-injury, no tongue biting, no sphincter incompetence, and no true unconsciousness. The eyelids are tightly closed, the pupils react, and the movements are theatrical. *Astasia-abasia*, inability to walk or stand but ability to move the limbs freely in bed, may follow slight injuries, fright or confinement to bed for a few days. *Paralysis* may be local or general and take the form of paraplegia, monoplegia or hemiplegia. Monoplegia of a leg is the most common. The knee jerk may be absent. The face generally escapes. It may be combined with rigidity or flaccidity, a little wasting from disuse, and possibly mechanical œdema in a dependent limb. *Contractures* are rare under puberty. They are usually more painful and more extreme than in organic disease, and relax during sleep and under anæsthesia. They may simulate torticollis, cervical caries, scoliosis or hip disease. The possibility of early disease in hysterical subjects must not be overlooked. The plantar, pharyngeal and superficial reflexes may be lost. The plantar reflexes are never extensor, and the deep reflexes are normal or exaggerated. There is no ankleclonus.

*Respiratory* symptoms may take the form of continuous yawning, slow and deep breathing, dyspnœa, orthopnœa, and barking or spasmodic cough. Violent stridor and asthmatic attacks may occur, if the child has had such symptoms before from organic disease. The *circulatory* symptoms are palpitations, tachycardia, abnormal pulsation in the abdominal aorta and carotids, exaggerated flushing and pallor, and dead fingers.

Symptoms referable to the *digestive* tract include anorexia nervosa, polydipsia, vomiting, cardiospasm, constipation, persistent diarrhœa, fæcal incontinence, prolapsus recti, hæmatemesis, enterospasm, and meteorism due to swallowing air or spasm of the diaphragm.

The *urinary* symptoms include anuria, pollakiuria, polyuria, enuresis, retention, and possibly albuminuria and hæmaturia. The *skin* may show vasomotor irritability, and attacks of pallor or flushing, the pallor being due to sudden dilatation of the splanchnic area; fugitive patches of congestion, dermatographia acroparæsthesiæ and erythromelalgia; and exudative skin eruptions, e.g. urticaria, local circumscribed œdema, angioneurotic œdema, urticaria factitia, i.e. produced by pressure, and serous or hæmorrhagic effusions. Almost any symptom of organic disease may be noted.

*Diagnosis.*—It is chiefly important not to overlook organic disease. Hysteria is comparatively infrequent in children, so it is advisable to be cautious in attributing symptoms to this cause. Occasionally supposed hysterical symptoms precede by years the evidence of organic mischief, for instance cerebral tumour.

The *prognosis* is favourable as regards the particular symptom and the general psychological state, provided it is recognised early and properly treated. Otherwise it is bad, for the affection becomes rooted in the child's inner



consciousness. The outlook is most favourable in the imitation hysteria which breaks out in schools.

*Treatment.*—In all the above conditions the treatment is both moral and physical. Separate the child from neurotic parents or attendants. Encourage it to exercise will and self-control. Auto-suggestion, as in christian science, is of great value. Direct education to bridling the passions, attaching little importance to trivialities, and taking a large view of life. Cultivate unselfishness. Provide continuous occupation to prevent introspection and self-indulgence. Understanding, tact, sympathy and firm moral support must be afforded by the physician. Intentional neglect is advisable in convulsive affections, and indeed for most symptoms. Point out the baselessness of phobias and obsessions.

Improve the nutrition and tone, and reduce the irritability, of the nervous system by rest, abundance of sleep, isolation, diet, massage, electricity, hydrotherapy, exercise and hypnotism. Removal from the old surroundings is necessary for treatment by intentional neglect. Choose a sanatorium in the country by preference. Keep the child in bed at first. Allow no alcohol. Gavage or the passage of a stomach tube may be needed for anorexia nervosa. Faradisation of the abdomen is beneficial in psychic diarrhoea, in which the stools are normal and frequent. Static electricity is best; galvanism for rigidity. Measures which produce a strong suggestive action through pain, unpleasantness or mystery, are the most useful, e.g. cold douching, gavage, galvanism, and the faradic current in aphonia and deaf-mutism. Hypnotism is a last resort for the worst cases. It is of doubtful value in hysteria and neurasthenia, and useless in psychasthenia. Drug treatment consists in tonics for the general health; bromides and hypnotics for sleeplessness; laudanum and purgatives for mental anxiety and depression; apomorphine for convulsions; trinitrin, pilocarpin and erythrol tetranitrate as vaso-dilators; ergot as a vaso-constrictor; calcium salts for eruptions; and assafoetida and valerian.

**Mental Deficiency.**—Backwardness, feeble-mindedness, imbecility and idiocy are merely differences in degree of mental change. A backward child has the mental condition of one younger in age. An idiot cannot attend and does not react to impressions from without. In imbecility the attention can be attracted but not fixed. In a small number the parents are normal and the child merely presents deviations from the normal. In the remainder one or both parents are insane, feeble-minded or physically degenerate. Consanguinity is of little importance unless there are predisposing causes in the family. The slighter defects, preventing the child being taught as a normal child, are present in about 1 per cent. of all children.

*Etiology.*—In about one-third there is a family history of idiocy, imbecility or some form of insanity; occasionally a history of intemperance or immorality, alone or associated with a low degree of intelligence,



congenital syphilis or tuberculosis. Nine-tenths of the cases depend on modifications of the germ cell or early embryo. Sometimes the hereditary defect only affects the male children. The influence of the age of the parents is shown in Mongolism. The older the mother, the more likely is mental defect to be present in the child or to follow some exciting cause, especially an infectious fever. Cretinism depends on athyroidea. In Mongols and microcephalus there are morphological anomalies but no gross cerebral destruction. Hydrocephalus only causes mental changes in late stages. Idiocy "by deprivation" is due to adenoids and deafness, or to ocular causes such as congenital cataract, hypermetropia and astigmatism. Paralytic idiocy depends on gross cerebral mischief. It is associated with paralyses and contractures, and includes many epileptic, paralytic, syphilitic and traumatic cases. Recurrent fits cause mental deterioration and usually depend on past encephalitis, syphilis or birth injury. The various cerebral scleroses, including family amaurotic idiocy, are endogenous or depend on injury at birth or encephalitis. Possibly the toxins of infective disorders, gastro-enteritis, chorea and rheumatism are exciting causes, independently of encephalitis. Malnutrition is a potent predisposing factor. Post-natal traumatism, heat, opium, malaria, fright and shock are rare causes, unless other factors are present. In most cases there is some primary constitutional weakness as well as an exciting factor.

According to Macpherson (1904) the absence of certain mental faculties is "wholly or in part correlated with the development of the physical organisation, especially that of the central nervous system, and it is rare to meet with imperfect structure of the nervous system in the absence of bodily imperfections." The greater the mental defect, the more pronounced are the malformations.

*Stigmata of degeneration*, or physical defects of other parts, are often present. Similar conditions are found less often in normal children, so too much stress must not be laid upon them. They merely afford presumptive evidence. The head may be micro-, macro-, hydro-, brachy- or dolichocephalic; asymmetrical; present an abnormally narrow frontal region, poor or absent frontal eminences; or show irregular bosses of syphilitic or rachitic origin. The face is asymmetrical. The ears may be unduly large and prominent, have adherent lobules, or show irregularity of the helix. A highly-arched or V-shaped palate is found in about half the cases, and in only one-fourth of normal children. Up to the time of the second dentition there is no difference between the palates of the sane and of idiots. Mentally defective children may retain the infantile form of palate up to the fifteenth year. Hence a deformed palate can only be regarded as one of a number of indications of imperfect anatomical development. A normal palate does not prove absence of degeneracy, nor does an abnormal one indicate its presence. Other anatomical stigmata are deformed nasal bones, an epicanthic fold, opacities in the media, high errors of refraction, badly developed



jaws and teeth, prognathism, narrow naso-pharynx, hare-lip, transversely fissured tongue, webbed fingers and toes, supernumerary digits, sluggish circulation, congenital morbus cordis and early puberty.

There is delay in acquiring the erect posture, walking, manual dexterity, speech and control over the bladder.

*Abnormal nerve signs* are noted on examination. The facial expression is lacking in intelligence, the mouth open and the forehead wrinkled. The tone of the facial muscles is poor, and there is defective capacity to fix the eyes and attention. The limbs lack control and co-ordination, the fingers twitch, and there is constant fidgets (microkinesis). The response to stimuli is slow. Tremors, athetosis, spasticity and contractures indicate organic cerebral mischief.

Various bad habits or *stereotypia*, such as pica, sucking, licking, tongue-sucking, lip-picking, nail-biting, head-scratching, teeth-grinding, head-banging, etc., fæcal incontinence, enuresis and masturbation may be present. They are signs of nerve-instability, except in some cases when they are clearly due to local or reflex causes. Nerve-storms, of the type described under "psychasthenia," may occur, notably inexplicable attacks of screaming or violent temper, and obsessions, morbid fears or moral insanity may be present. In quite one-third the heart's action is feeble, and there may be bradycardia. It is more often enlarged than undersized. Murmurs, especially mitral ones, are not uncommon and the pulse may be irregular. These children are more often delicate and liable to tuberculosis than normal children. This is partly due to defective innervation and circulation, and partly to less care by others and less competence to look after themselves.

*Diagnosis.*—Many dull children show none of the above defects and many who do show them are by no means feeble-minded. In infancy the diagnosis is very difficult. The face is a better mirror of the mind than in adults. Its expression may arouse suspicion. It may be stupid, fatuous, vacant, sullen or apathetic, or the child may smile inanely, grin monotonously and make grimaces. The babe may recognise neither mother nor nurse. Lax jaws, pendulous lips and slobbering are suspicious signs. An exceptionally "good," placid baby, vacantly smiling and rarely or never crying, is generally feeble-minded.

In the presence of good physical development, without deformity or contracture, try intellectual and physical tests, note the attitudes and demeanour, enquire as to conduct and mode of life, the date at which the various functions were acquired, and compare the power of reading, writing and doing arithmetic with that of normal children. Note the facial aspect, and the amount and duration of attention paid to various objects and stimuli. Note the presence of undue self-consciousness, nervousness or fright, or unusual lack of shyness. Test the visual and auditory reactions. If they are normal, there is often only a mild degree of



idiocy. Test sensation to pain ; to heat and cold ; and to taste, by a 2 per cent. solution of saccharin, quinine 2 per cent., HCl 2 per cent. and common salt 5 per cent., noting the effect on the facial and oro-lingual expression. All these reactions may be modified by intercurrent disease. Note speech defects and examine the reflexes.

*Prognosis.*—If nutrition is poor, do not give a definite diagnosis or prognosis until it improves. Physical weakness and lack of resisting power are common, especially in Mongols and cretins. Quite 50 per cent. die between 10 and 30 years, and few live over 40 years of age. In severe forms the child is unable to recognise its parents, know its own clothes, find its way about or feed itself. Abstract ideas of duty, etc., are absent. Emotional faculties are fully developed. The outlook as regards mental development varies with the type and degree. Often children most normal in appearance develop the worst ; so, too, those who have fits. Mongols can rarely do much brain work. Cretins develop well, if put under thyroid treatment from the first year. Organic heart disease is an important factor in causing continued deterioration. It is impossible to be certain how much improvement will take place in any particular case. Much depends on education.

*Training.*—The mental processes are slow and difficult to control, and the mental impressions are less powerfully fixed and less co-ordinated for future use. Teaching must be objective. Cultivate attention through the sight and by this means train the child in the control of the eye muscles. The muscular sense is educated through finger exercises, movements of the hands and arms, and in estimating the weight, size and shape of objects. Other muscles of the body must be also trained. Differences in temperature, weight of coins, sounds and colours are useful exercises. Later, teach the child to imitate the movements of the teacher in writing and speaking. Then pass on to drawing, modelling, games involving counting and calculation or the sense of touch and accuracy of sight, the value of coins, marching and other forms of drill. Encourage self-help in all forms. Teach reading and the knowledge of figures and time. In early stages limit education to one sense at a time, viz. sight, touch and hearing successively. Over-exercise is injurious to backward and imbecile children because of the heart weakness. Amusements and simple entertainments are necessary. The teacher must live with the child. No school work is permissible before 7 years of age. Development generally stops at 16 years. Attend to any physical troubles, such as adenoids, eye defects, rickets, contractures and deformities. If there is any suspicion of cretinism, give thyroid extract. If there is no cretinism, it produces no improvement and thyroidism soon ensues. Training of muscles and brain must be steady and persistent.

**Mongolian Idiocy.**—This is a variety of congenital idiocy in which the features are of the Mongol or Kalmuck type. The appearance of the eyes



is like that of the Chinese and Japanese. From 3-5 per cent. of all idiots are Mongols and they present a remarkable likeness to each other.

*Etiology.*—A Mongolian idiot is usually the last child of a large and normal family, as if due to exhaustion of the reproductive power. Generally the mother is of advanced age. Occasionally it is the product of the union of an old man with a young woman. In rare instances it is the first child of young parents. Exceptionally a later child may be normal. One or both of twins may be affected. Mongolism is more frequent in Great Britain than elsewhere. It is doubtful whether it occurs among the Jews. The causation is obscure. Consanguinity, ill-health and syphilis cannot be regarded as more than encouraging factors. Sutherland obtained a family history of syphilis in 14 out of 25 cases but anti-syphilitic remedies are useless. The cerebellum, pons and bulb were unduly small in 5 cases examined by A. W. Wilmarth. Oberther (1904) found meningo-encephalitis in 3 cases, with atrophy and chromatolysis of cells. W. B. Hill (1908) noted thinning of the cortex and rarefaction of nerve cells, especially the large pyramidal cells. Some of these effects may be sequels. One of Oberther's patients was 12 years old and died from tuberculosis.

*Symptoms.*—At birth the child is small. Growth is slow and at 1 year the child looks like a 6-month old baby. The stature is always subnormal and the weight about one-fourth of the normal. The head is small, brachycephalic, the antero-posterior diameter being equal to the transverse, and the occiput much flattened. This has been ascribed to premature ossification of the basilar bones or defective growth of the structures at the base of the brain. It is probably secondary to lack of development. The fontanelles and sutures close late. The hair is fine and abundant. The face is small and rounded; the cheeks round, soft and chubby, of a dirty yellow or ruddy colour, and often show venous congestion. The cartilaginous part of the nose is broad, the alæ extruded and opening in an upward direction, the nasal bridge flat or sunken, and the tip frequently red and retroussé. The ears may be unduly small. The palpebral apertures are almond-shaped, small, narrow and oblique, sloping upward and outward like the orbits. Marked epicanthic folds are due to redundancy of skin and give an appearance of increased distance between the oval, tilted eyes. Chronic blepharitis is common. Nystagmus and squint are often present during the first 6 months, possibly due to refractive errors. There is no puckering of the brows, no puffiness under the eyes, and no fat pads above the clavicles as seen in cretinism.

The lips are normal, the palate often high and narrow, the mouth small and usually kept open, and the naso-pharynx small. Apparent enlargement of the tongue at birth is either due to abnormal shortness of the mouth, causing a normal tongue to protrude, or there may be actual enlargement of the tongue. Later on it becomes curiously rough and fissured transversely,



raw and sodden, with swollen papillæ. J. Thomson has carefully observed the progress in these cases and states that the swelling of the papillæ begins in the third to ninth, occasionally in the second month, and the fissuring in the second to sixth year. He ascribes the condition to deficient resistance of the lingual mucosa and to tongue-sucking, present in 59 out of 69 cases and possibly overlooked in others. The upper lip is often sucked in. The character of the tongue depends on the degree and duration of tongue-sucking. Its protrusion is not constant as in cretinism and may be absent during sleep. Dentition is delayed and the teeth ill-formed, perhaps widely separated. The voice is guttural and the child is apt to crow as in laryngo-spasm.

The hands are broad and square, with short, squat, blunt fingers. The thumbs and little fingers are abnormally short. The second phalanx of the little finger is unduly small and the finger incurved laterally. The fingers may be spread out widely, the *main en trident*. The ligaments are loose, and the mobility of the joints may be so great that the fingers can be bent back to touch the arm. The muscles are hypotonic. The skin is smooth and white in infancy and furfuraceous later. The trunk is not dwarfed. The abdomen is generally large and distended, and may show umbilical hernia. The genital organs are small and cryptorchism is sometimes present. The pelvic and long bones are smaller than normal, and the bones show delayed ossification. The limbs may be short and their ligaments unduly lax. The feet are flat and broad. Muscular power is feeble and walking delayed. The extremities are blue, cold and clammy, for the circulation is feeble. Sitting cross-legged is a favourite attitude. Knee jerks are often absent and the plantar reflex extensor. Constipation is common and the habits may be quite cleanly.

Congenital morbus cordis and other defects are present in 20 per cent. (Sutherland). Garrod (1898) drew attention to the frequency of the heart mischief. Hutchison found cardiac defects, often of the septum, in 5 out of 18 cases. Other deformities are spina bifida, club-foot, imperforate anus and renal cysts. Mentally there is nothing noticeable beyond backwardness, less marked than in cretins. They are "good" babies, giving less trouble than normal ones, apathetic, easy to manage and easily amused, and will lie for hours smiling placidly, babbling unintelligently, grimacing, making curious throaty noises, and biting the fingers. As they grow older they may be bright and lively, fond of gestures, music and dancing, and imitative. The expression is fatuous in bad cases. They are liable to stammer and have difficulty with the letter S. Sight and hearing are good. They are sometimes obstinate, rarely spiteful or mischievous, and never erotic or vicious.

*Prognosis.*—Most of them die under 3 years and almost all the rest before 20 years of age. Their vital power is small. They have a subnormal temperature, are very sensitive to cold and bear illness badly. Heart lesions increase the gravity. Death results from diarrhœa, bronchitis,



pneumonia, phthisis or other intercurrent disease. Phthisis may exist without cough or difficulty in breathing. If they live, they develop into affectionate imbeciles and are never able to look after themselves, much less earn a living. Muscular power and co-ordination are acquired very slowly. The patient cannot walk or talk before the sixth year. They are capable of a certain amount of education, but remain very backward and can rarely read more than a few short words.

*Treatment* is unavailing. A few have improved on thyroid medication. Training is the same as that suitable for the feeble-minded.

**Moral Insanity.**—Psychical defects have the same etiological foundations as mental deficiency, chiefly a family history of epilepsy, disorders of the intellect, or moral degeneracy as shown by excessive alcoholism and immorality. The child is often epileptic. In others the cause may be the action of toxins, injury or disease. There is a temporary or permanent defect of moral control, moral consciousness or will power. Lack of moral training is a potent cause in the delicate and crippled. The most common indication of imperfect moral control is uncontrollable passion on trivial provocation. Screaming attacks in infants may be of this nature but they are more often due to physical pain, a pin in the clothes, constipation or day-terrors. Next in frequency comes a nasty spitefulness or cruelty, shown by playing cruel tricks on other children, pinching, scratching, biting, banging, etc., and by cruelty to insects, birds and animals. Thus, a 9-year old girl had a propensity for trying to drown little boys, apparently from no motive save an innate instinct of murder. She had always been wilful, disobedient, mysterious in her ways, and mentally somewhat dull, and was unduly composed during her trial (*Daily Express*, 1903). The cruelty may take the form of trying to smother babies, inserting injurious articles into their mouths, or setting fire to the bedding. Pleasure is nearly invariably shown in the sufferings of the victim. A boy drowned a younger boy that he "might see the little devil kick in water." Pyromania is due to a love of seeing fire or a craze for excitement. It is common in epileptics. Cruelty may arise from jealousy and not be pathological.

Defective moral control is also shown by exaggerated jealousy, disobedience, and a reckless disregard for authority, parental, tutorial or legal, in spite of punishment. This condition leads to criminal acts and conflict with the law in later life. Dishonesty, stealing, destructiveness, wanton mischief, absence of shyness or reserve, shamelessness, immodesty leading to indecent exposure and open masturbation, sexual precocity, immorality and perversions, viciousness, habitual and useless lying and romancing are all at times present in different cases. The child rarely has a true conception of right and wrong, but may express fear of punishment and appreciate its effects. Sorrow is rarely felt or expressed for any misdeed.

All these symptoms can be ascribed to the animal desire for self-gratification independent of the feelings of others. A weak or defective



will is indicative of weak inhibitory powers, the normal state of infantile life. This is shown by the lack of control over the emotions of laughing and crying, comparable with the emotional state so often seen in chorea. Some of the signs are due to the persistence of an earlier normal state.

*Diagnosis.*—It is important to recognise that these psychical states may be quite independent of idiocy or disease, and due to delayed development. The normal state of a child depends on age, environment and heredity. At 5-6 years it should show consideration for others and at 8 years have definite ideas of right and wrong. In other cases there is a morbid loss of an already acquired normal psychical state. Untruthfulness may be due to timidity, a vivid imagination, the invasion of specific fevers, or the onset of organic cerebral mischief. Spitefulness, cruelty, lying and temper are all common in neurotic children and do not necessarily foreshadow criminal lunacy. Dishonesty is normal in young children for the gratification of appetite. Hysteria may lead to it from the desire to obtain gifts for some object of worship. The remark of a child "Does oo love Dod? Then oo shall see Dod," as he squashed a fly on the window-pane, can hardly be regarded as a symbol of moral obliquity; nor the administration of an enema to a toy elephant by a 5-year old girl as a sign of indecency. Moral delinquencies should be due to little or no provocation, persistent, and unassociated with ability to realise the wrongfulness of the action or with true penitence.

True moral defectives, without some stigmata of degeneration, form a very small group. Most of them are feeble-minded. If there is idiocy, the defective control and impairment of intellect vary directly as the degree of idiocy. Suggestive additional symptoms may be present, such as an abnormal desire for solitude, a lack of natural affection, an incapacity for sustained attention or a defective memory. Exaggerated emotions are due to defective inhibitory volition. Deficient moral consciousness may be shown by apparently complete ignorance of the bearings of the various acts, e.g. passion, violence, cruelty or indecency. Yet with all these the intellect may be normal. It must be noted that moral control does not vary with the intellectual capacity. Sometimes periods of defective control alternate with periods of a normal psychic state.

The *treatment* is similar to that of the weak-minded. Parents and guardians must be impressed with the risks of injury to self and others, the effects of indecency and immorality, and the liability to personal and family disgrace. The difficulties of education are great. These children must not be sent to school, for they are almost certain to be expelled. A new school or a change in surroundings is temporarily beneficial, but the effects are of short duration. The influence of bad literature, "penny dreadfuls," is potent for harm. Those most dangerous to others are the ones in whom moral insanity is combined with intellectual superiority.



High class criminals may be of this type and should be permanently segregated. There is always a possibility of improvement.

**Insanity.**—*Acute delirium* is the most common form of mental instability. It is screaming, muttering, or associated with terror, hallucinations, usually visual, and motor explosions. There is generally fever. *Mania* is rare, usually of short duration but apt to recur. Savage has reported it in a 3-year old boy. It takes the form of extreme elation, restlessness and variable fancies. Less often there are delusions, suicidal impulses and depression; or continuous weeping, melancholia and religious mania. A case of the former type, in a 5-year old girl, came under my notice as a sequel of epilepsy. Child-insanity is often associated with changed affections, obstinacy, anger, violence, unreasoning terror, hallucinations, blasphemous and indecent language, and even suicidal impulses. Complete rest and large doses of bromide are necessary. *Folie périodique* has been reported by Magnier in a girl, 12 years old, with suicidal tendencies. Eighty per cent. of the cases begin under 25 years.

*Acute mental confusion* may follow fright, injury, prolonged excitement and fever, notably typhoid. It is associated with delirium and stupor. The prodromal symptoms are restlessness, dulness, apathy, wet and dirty habits, and perhaps hallucinations. It is more allied to the delirium, which is liable to occur in the neurotic from slight fever, than to true insanity.

**Suicide** in children attending school is uncommon in Great Britain. Eulenburg (1907) states that there were 1152 cases in Prussia from 1880-1903, 340 in the higher schools and colleges. Mental and nervous troubles were present in 10 per cent.; 423 were ascribed to punishment, non-success in class work, or removal for misconduct; no cause was found in 265; some were due to love affairs and philosophical studies. It is most apt to occur in schools for backward children, from excess of theoretical work and lack of out-door exercise. Suicidal impulses arise in neurotic children from ennui, melancholy, disappointment, physical pain, a desire to attract attention or be regarded as of importance, and from ignorance or curiosity as to death and the future. They are rarely carried out beyond the extent of causing illness.

**Dementia Præcox** (Kraepelin), or acquired imbecility (Esquirol), begins in youth or early adult life. "Præcox" means that it is "untimely," before its time. Dementia is an unemotional state, one of pronounced inactivity. It implies full development of the mental powers and permanent loss, and is therefore an acquired disease, differing from "amentia" or congenital enfeeblement. The characteristic feature is loss of memory. On the whole "*Adolescent Insanity*" is a better term and it may be divided into hebephrenia, katatonia and paranoia, sub-divisions which are closely allied and overlap. Dementia præcox is an unsatisfactory title



or there is not always permanent loss, some recover, and the loss of memory is not invariably a prominent feature.

*Hebephrenia* begins with hallucinations and delusions which tend to disappear. The chief symptoms are eccentricity, jerky mannerisms, loss of voluntary attention and activity, loss of interest, apathy, perhaps mutism, and sometimes maniacal excitement, followed by depression and permanent mental debility.

In *katatonia* there are hallucinations, apathy, loss of interest and attention, stereotyped movements, and mutism, merging into katatonic stupor and muscular tension. *Paranoia* begins with unjustified suspicions, rapid development of unsystematised delusions, hallucinations and delusions of persecution and grandeur, which tend to pass off as the mental state deteriorates. Some katatonic stupor, mannerisms and stereotypy may be noted. The premonitory signs of this form of insanity may take the form of simple mental deterioration, a few indefinite delusions, neurasthenic or hysterical symptoms. Sometimes there are abnormal fears, great indecision, and destructive or criminal impulses. At puberty these children may show great exaltation or depression of spirits, with intellectual precocity. In a few months to 2 years signs of mental dulness and failure are evident.

*The early symptoms of mental disorders* are:—hyperæsthesia, anæsthesia, hyperacusis; insomnia; disorders of perception, viz. illusions, hallucinations and imperception; imperative ideas; disorders of emotion, memory and conduct; disorders of instinct, viz. lack of interest, excess of energy, extravagance, boastfulness, excessive eating, sexuality and the collecting mania; and delusions. Fortunately mental disorders are comparatively rare in children, except in the degenerate class of mental defectives and in epileptics. Nevertheless the infrequency of their occurrence is an insufficient ground for omitting reference to them in a work of this nature, for they are liable to be overlooked in the early stages. Unfortunately treatment is rarely beneficial, except in acute cases in which chief reliance must be placed on isolation, rest, liberal diet and nursing. The recognition of the frequency of moral insanity and the dangers of overstimulation of the mental faculties of children, especially if neurotic or mentally defective, should be constantly borne in mind by those in whose charge they are placed for educational purposes.

*Menstrual psychoses* take the form of loquacity, incessant talking and singing, agitation, and hallucinations of sight and hearing. They are of the same nature as adolescent insanity, the attacks occurring during menstruation and subsiding soon after, but liable to recur.

**Speech Defects.**—Wyllie (1892) divides the stages of acquirement of speech into:—(1) Inarticulate sounds, at first instinctive; crying, laughing and grunting; (2) Facial expression and gestures; (3) Babbling and crowing, mimic reading, and echolalia or “parrot talk” at the end of the



first year; (4) Intelligent speech. Girls speak 2-3 months before boys. *Delayed speech* is due to mental defects, backwardness from rickets or acute illness, or defective hearing. Some children learn much later than others, without there being any abnormality. Delay beyond the third year of life is suspicious, and such children must be examined for physical or mental defect. Yet occasionally speech is not acquired until the fifth to seventh year and is quite normal in later life. Retarded speech delays mental development.

The *speech centre* is supposed to be in the posterior third of the third left frontal convolution (Broca). It is bilateral at birth. The left one normally develops, but if it is damaged or destroyed in early life, the right centre takes on its functions. Aphasia in the first 4 years of life is soon recovered from. Wernicke's region consists of the angular gyrus, the supramarginal gyrus, and the posterior parts of the first two temporo-sphenoidal convolutions. Obliteration of the middle cerebral artery causes destruction of both these regions, so the types of aphasia vary with the extent and localisation of the obstruction. Marie (1906) argues that Wernicke's region is the true speech centre; and that Broca's region has nothing to do with speech, can undergo softening without causing aphasia, and may be quite unaffected in typical cases. Although it is affected in 50 per cent., it is because the damage is due to obstruction to the middle cerebral artery.

*True aphasia* implies a defect in the ability to understand language. In the *motor aphasia* of Broca, the lesion being in Broca's convolution, the patient cannot speak but can understand. In the *sensory aphasia* of Wernicke, the lesion being in Wernicke's region, the patient speaks more or less incoherently but cannot understand speech (word-deafness) or written symbols (word-blindness). Marie maintains that the only difference in these two varieties is the absence of the power of speech and that this is due to anarthria, from damage to the region of the lenticular nucleus. In both there is inability to read, write, and understand complicated questions. Wernicke's centre is an intellectual and not merely a sensory centre. The apparent word-deafness is due to lack of comprehension, not to a damaged auditory centre.

*Anarthria* is a true motor aphasia in which the child cannot speak or is only able to utter unintelligible sounds, although able to understand, read and write. The lesion causes inco-ordination and is subcortical, in or near the lenticular nucleus or internal capsule.

*Amnesic aphasia* is the loss of memory for words which may result from severe illness. The loss depends on deficiency of ideas. In chorea this loss may be partly motor. It can be called "*functional aphasia*," and may follow severe fright. *Paraphasia* is the use of words incorrectly or without meaning. Rarely the speech defect depends on congenital



absence of the speech centres or on bilateral damage of the motor areas for the lips and tongue by meningeal hæmorrhage.

**Congenital Word-Deafness** is very rare, independent of deafness and mental defect, and does not always affect musical sounds. It is due to some defect in the auditory centre, which is said to be in the superior temporo-sphenoidal convolution, though Marie can find no clinical or pathological evidence in favour of this site. The most recent view of the site of the auditory centre (Flechsigg, 1908) places it mainly in the anterior transverse temporal gyrus (Heschl's convolution) on the upper or Sylvian surface of the temporal lobe, and continuous with the upper temporal convolution. In a girl, 11 years old (Macleod Yearsley), there was only vowel hearing and echolalia. She was intelligent and could lip-read. Later the echolalic motor response was necessary to enable her to understand but subsequently a slow reply was obtained without.

**Congenital Word-Blindness** is more frequent in boys than girls, and in the lower classes, and often familial. S. Stephenson found 5 females and 1 male affected in 3 generations of a family. It was transmitted from the second to the third generation by an unaffected female. C. J. Thomas (1905) estimated that it was present to a considerable degree in 1 in 2000 London elementary school children. The first case was described by Pringle Morgan (1896). Kussmaul (1877) used and explained the term in reference to acquired cases due to disease of the left angular gyrus. Slight degrees are apt to be overlooked in hospital work. The affection is rarely recognised under 7 years of age.

It is a congenital defect in the visual memory centre for words and letters, or a failure in co-ordinating written and printed symbols with the sounds and ideas which they represent. There is an imperfect relationship between the visual, auditory and articulatory centres. It is a word and letter blindness, an inability to read (developmental or congenital *alexia*), and often associated with a variable amount of word-deafness.

These children are intelligent, and simply have a variable degree of difficulty in learning their letters and in reading either printed or written words. Often there is no difficulty with figures and rarely the reverse condition. A few of them can understand on reading, but are unable to read aloud, that is, there is no true word-blindness but a motor failure, not in speech but in translating visual characters into audible sounds. Ordinarily the visual images of words fail to be stored up. The difficulty is usually ascribed to stupidity, mental defect or error of sight. Visual acuteness and the fields of vision are normal; hypermetropia is common. Memory is good and the boy may be able to play games well. One child was brilliant and became an excellent surgeon. These children do not read for pleasure. Some can read slowly. Recognition of arabic numerals is easily acquired. They can be taught orally and remember words by sound.



*Diagnosis* is easy. Defective vision can be excluded, for the child cannot read large type any better than small. Words can be recognised by spelling them out. An error of refraction may lead to the defect being overlooked. Try if the child can understand spoken words and remember them. Spontaneous speech is usually limited.

*Treatment*.—The earlier the diagnosis is made and special methods of education adopted, the better and quicker is the improvement. Increase the child's vocabulary by placing him under conditions where he hears plenty of talking, reading and word-building. Constant repetition of visual impressions is needed, so lessons must be short and frequent. The child cannot be taught in class for it is soon discouraged. Probably the right angular gyrus takes up the function.

**Deaf-Mutism**.—A deaf-mute is not necessarily totally deaf and dumb. Quite half of them have some hearing power, though not enough to enable them to acquire speech. Some are congenitally deaf. About half are cases of acquired deafness, complete or incomplete. A few have congenital word-deafness. Bezold states that absolute deafness is more common in acquired than congenital cases. If it occurs in infancy, it may be erroneously ascribed to a congenital defect. A deaf child is necessarily backward. Some hear a little on one side.

Heredity exerts an influence to the extent that the affection is most common in families liable to "hardness of hearing." Deaf-mutes do not transmit the condition to their children. Consanguinity and debility in the parents are possible causes. Bond (1905) pointed out that deaf-mutism commonly affects children of the same sex, male or female, in the particular family. In other instances the children are twins or contiguous births. Labyrinthine anomalies of embryonal origin have been found in several cases.

Deaf-mutism may develop before or after speech has been acquired, and may occur as late as the sixth year. After that speech is retained though deafness arises. It is due to ear mischief, often secondary to adenoids and throat affections, nervous diseases and injury. Some result from labyrinthitis hyperplastica of syphilitic origin but the bulk are due to labyrinthitis exudativa, secondary to scarlatina, measles, other infections, and meningitis. Otitis media may also be present in either variety. Rarely it is due to prolonged impaction of wax.

*Diagnosis*.—A normal child hears in the second month. Deafness cannot be recognised with certainty until 4-6 months of age, perhaps not until 1 year. If there is mild idiocy, it is difficult to differentiate the dumbness from that of deafness.

*Treatment*.—Prevention consists in care of the throat during infective disorders. Mercury, pot. iod., and pilocarpine may be useful in labyrinthitis. Once the affection is fully acquired the child should be taught



*lip-reading.* Teaching must be begun early, continued for 6-8 years, and requires great perseverance. After this the child can be taught quite well with ordinary children, if he can see the teacher's face. The *sign and manual* system takes 5 years and is not nearly as satisfactory. It is a hindrance to proper education by the oral system. Lip-reading is better for intelligent children and indeed for any child capable of being taught at all. For incomplete deafness ear trumpets and other appliances, and special schools and teachers are required.

**Idioglossia** is a defect of hearing, analogous to errors of refraction in the eye, or a variety of word-deafness, or a defect of articulation. The child is voluble, though unintelligible, and talks a gibberish of its own. It is intelligent, not deaf, has no local defect, and can read and write normally. Similar speech has been noted in Mexicans, Italians and South Sea Islanders. It is sometimes familial and associated with a family history of insanity. Occasionally there is a defect in visual and musical sense, and in the power of concentrating attention.

Formerly these children were mistaken for deaf-mutes. They hear well and express themselves by articulate sounds, but the sounds are unlike those of any known language, and their speech is distorted or imperfectly pronounced. The same sound is always used by the same child to express the same word. The speech is somewhat like "baby" English and may be due to imperfect training. In consequence of inability to utter certain consonants, others, usually T, D or N, are substituted. It must be treated by oral methods as in deaf-mutism. Normal speech is almost always acquired.

*Lalling* is the persistence of infantile speech, usually due to defective intelligence. It is an aggravated form of lisp. Consonants may be transposed and syllables or words dropped.

*Lisping* is a faulty articulation of certain consonants or substitution of wrong consonants, the result of local defects or malposition of the various organs employed in articulation. It is normal in infancy and may persist from faulty habit or imperfect education; or may begin when the milk teeth are shed and become habitual. The commonest form is the use of Th for S. In paralysis of the lips the labial consonants P, B, and M are pronounced as F and V; in palsy of the tongue the linguals L and S are said as Y and Th; in palatal palsy M replaces P and B. In hare-lip there is difficulty with M, P, B, F and W, letters absent in the words of various American tribes. In cleft-palate the voice is nasal and there is difficulty with dental and palatal sounds, especially T, D, S, R, L, K, G. A high palate may have some effect. Frequently W or L is used for R; sometimes T for G or K; and L may be replaced by D, T, S, J, N, Ng.

**Stammering and Stuttering.**—Speech depends on the co-ordinated action of three mechanisms under the control of the nervous system. The expiratory muscles supply the blast of air. The laryngeal or phonetic



system produces vocalisation by approximation and tension of the vocal cords. The oral, buccal or articulatory mechanism consists of the lips, tongue and soft palate, and varies the size and shape of the orifice through which the air passes and produces words. The vocal and oral mechanisms can be used separately but are co-ordinated in speech. Any one of these parts may be at fault. As a rule the defect is in the management of expiration or delay in laryngeal action. Thus stammering may be due to spasm of the respiratory muscles or diaphragm, failure of articulation, or failure of co-ordination, i.e. of nervous control. It may be defined as an irregularly intermittent fault of speech, a spasmodic neurosis. It is a want of promptitude in vocalisation, a sudden check in utterance, or rapid repetition of consonantal or vowel sounds.

*Etiology.*—Macleod Yearsley (1907) in a series of 800 cases found the percentage causation was heredity 40, association and mimicry 29, shock and fright 16, illness 15. Congenital cases are rare. They are due to weakness of the syllabic co-ordinating apparatus, defects in the air passages or in breathing. It is uncommon among imbeciles and not a sign of weak-mindedness. Temporary cases occur during teething, pertussis, etc. If caused by fright, it may be choreic in character. Faulty education, imitation and habit induce or maintain it.

A neurotic family history is of prime import. It affects 4 boys to 1 girl. It is rare before the third year of life ; except in a mild and temporary form until speech is properly acquired. Three-fourths of the cases begin between 4 and 9 years of age, and 10 per cent. after 12 years. It is a serious disability, a handicap in most walks of life, and an annoyance to self and others.

*Varieties.*—The simplest variety is a difficulty in articulating an initial consonant or a spasmodic arrest before some syllable. The arrest may be complete, due to spasmodic arrest of the diaphragm and cessation of breathing, and analogous to the difficulty of utterance during violent passion. Usually it affects the explosives, which are not vocalised at the proper time. Or the initial consonant, fricative or nasal resonant, is prolonged because the breath is driven out feebly and inefficiently. Or there may be stuttering, the spasmodic repetition of the same syllable, with shallow and badly managed breathing, low and hurried speech, clipped and slovenly articulation. More severe types are associated with :—(1) Sounds, e.g. inarticulate whimpering noises, grunting, whooping, crowing or interpolated meaningless words ; (2) Movements, e.g. grimaces or severe facial contortions, fidgets of hands or feet, motor spasm of limbs, biting lips or tongue, slapping, pinching, kicking or stamping. The voice is laboured and the secretion of saliva excessive.

*Physiological Alphabet* (Wyllie).—Vowels—i, e, a, o, u are pronounced in the latin manner as ee, eh, ah, oh, oo ; and y as an initial is closely allied



to i, and w to u. The voiceless oral consonants are purely buccal, the others contain a vocal element.

	Voiceless Oral.	Voiced Oral.	Voiced Nasal.
Labials .. ..	P	B, W	M
Labio-Dentals ..	F	V	
Linguo-Dentals ..	S	Z	
Ant. Linguo-Palatals	Sh, T	D, L, R	N
Post. Linguo-Palatals	K, H, Ch	G, Y	Ng

In this alphabet C = S or K; Q = Kw; X = Ks. Usually in stammering the explosive or closure consonants B, P, D, T, K, hard G, are affected, due to spasmodic closure of parts of the air passages which should be thrown open. Or the fricatives, the continuous consonants, V, F, C, S, Sh, W, M, N, are affected, through fixation of the parts engaged in uttering the particular sound. Or the vowel sounds are at fault, from mismanagement of breathing or glottic spasm, a condition followed by rapid speech until breathless.

Every letter is partly laryngeal and partly oral, the vowels being more purely laryngeal than the consonants. In closure of the lips, first stop position, the letters P, B and M can be uttered. With the tip of the tongue applied to the hard palate behind the upper teeth, second stop position, T, D and N are formed. In the third stop position, approximation of the back of the tongue to the soft and hard palate, K, G and Ng (in wing) can be pronounced. In spasm of the true and false vocal cords, a fourth stop position, vowel sounds are inaudible. The labio-dental and linguo-dental are also stop positions. In uttering nasal resonants, voiced nasal consonants, the soft palate drops and air escapes through the nose. If there is nasal obstruction, M is pronounced like B.

*Diagnosis.*—Examine the child by means of the test sentences recommended by Wyllie. They represent the usual order of difficulty :—

Vowels.—Eels ail amid ocean ooze.

Voiced Fricatives.—We visit the Zulus like ramblers yearly.

Voiceless Consonants.—Far shores seem thinly hazy.

Nasal Resonants.—My nephew.

Voiced Explosives.—Best gold dust.

Voiceless Explosives.—Two poor comrades.

The difference between stammering and stuttering is more easily recognised than defined. A stammer is absent during singing and declaiming, and often when whispering. It is mainly a defect in co-ordination of the respiratory and laryngeal mechanisms, with inability to produce articulate sounds. Sometimes there is a malformation of the speech organs. The eyes may roll and the features be distorted. An abnormal mental and emotional state depends on lack of co-ordination of mental and vocal



processes. There is rarely any spasmodic or convulsive action of the vocal apparatus.

Stuttering is cerebral in origin and oral in production. There is lack of nervous control, an abnormal mental and emotional condition which is primary and combined with improper respiration and vocalisation. This induces vicious attempts at utterance, and painful and rapid repetition of the initial syllable. Inability to produce articulate sounds is rare. With stuttering there is associated an overflow of the cerebral stimulus to the larynx, etc., causing congestion of the face, cyanosis, spasmodic and abnormal breathing, and excited physical movements. Van Praagh states that a stammerer does not know *how*, a stutterer does not know *when* to speak.

*Prognosis.*—The outlook is bad, if the intellect is defective. It depends on the intelligence and co-operation of the patient. It is worse, if associated with severe spasmodic movements. Mild cases frequently get well after puberty.

*Treatment.*—In teaching children to speak they should be made gently to repeat letters and words, until they do so without stumbling. Attend to the general health and guard against neurotic tendencies. Encourage self-confidence. Teach breathing exercises and see that the air passages are unobstructed by adenoids, enlarged tonsils or nasal defect. Attend to any defect in the mouth.

If there is the least sign of stammering begin early a regular and rigid system of training, after the onset of the second dentition, although many recover spontaneously. The best results are obtained at 12-16 years of age because the child can appreciate the method and understand its importance. Throughout life the patient, in bad cases, must attend to voice production for relapse is common.

First teach proper breathing. Encourage abdominal respiration with attention consciously directed to it, "diaphragmatic drill." Then teach prolonged utterance of vowel sounds, aloud and in whispers; words of one syllable beginning with (a) vowel, (b) consonant; words of two syllables and more; poetry, prose and declamation. The child should vocalise strongly, without drawing in breath, for this may lead to inspiratory or "draw back" phonation. If there is the least difficulty, he should stop and not make repeated attempts to overcome spasm. Any difficult letter or sound must be practised separately and all consonants clearly enunciated. Speaking must not be attempted until the lungs are fully expanded. The patient should keep a pebble under the tongue, to prevent it cleaving to the floor of the mouth, or a piece of wood between the teeth, and speak slowly and distinctly, in a loud full voice, prolonging the vowel sounds and beating the time of the syllables with one hand. Attention must be directed first to vocalisation and then to articulation. Next practise reading aloud, poetry first for it requires more voice, in a large room, in a distant room with the intervening door open or in the open air, without shouting or



allowing the voice to drop for this shows that the chest is not kept full. Underline defective words and try them separately. Intoning, declamation and singing are often curative, but may induce a sing-song delivery. Guard against nervousness and excitement. When the child is old enough he should have exercises in which his attention is directed to the mechanical movements in voice production, e.g. the exact position of the tongue and lips. He can then correct his stammer by placing his mechanism in proper order and thus overcome the hesitation.

These children must not go to school. Bad cases must be sent from home and put under the charge of an experienced teacher, best of all under single charge and not with others similarly affected. Treatment is individual and little progress can be seen for a month or more.

Yearsley recommends a course of 4 hours daily, 6 days a week, for 6 weeks. It is directed to the cultivation of self-possession, efficient breathing and proper vocalisation. Respiratory exercises teach thoracic, diaphragmatic and abdominal breathing. Vocal gymnastics are directed to practising the natural articulation of vowels, explosive utterance, rising and falling inflection, and prolonged articulation at normal tone. The third exercise is devoted to combined vocalisation, breathing, and physical drill with dumb-bells at 80 movements per minute. Finally the child reads and recites in company, syllable by syllable. Half-an-hour, morning and afternoon, is devoted to each exercise. It requires 2 weeks to re-establish normal use of the speech organs; 2 weeks to encourage application and test ability; and 2 weeks for training to deliver written speeches and recite slowly and deliberately.



## CHAPTER LIX.

### THE MUSCLES, BONES AND JOINTS.

*Muscular Defects — Myositis — Myositis Ossificans — Torticollis — Lateral Curvature — Sprengel's Deformity — Club-foot — Osteomyelitis — Dislocations — Pulled Elbow — Arthritis — Rheumatoid Arthritis — Pulmonary Osteoarthopathy.*

*Congenital absence of muscles* may be partial or complete. It is most common in the pectorals, and next in the trapezius, serratus magnus and quadriceps extensor femoris. Absence of the ocular muscles may depend on infantile nuclear atrophy. Absence of the pectorals may be associated with absence of one or more ribs, aplasia of the sterno-mastoid and other muscles, atrophied skin, defective development of the breast, webbed fingers and webbing of the arm to the chest, and Sprengel's deformity. It is rarely bilateral. Permanent defect is not nearly as disabling as might be expected. Unilateral absence has been recorded in about 70 cases (J. G. Forbes, 1907). Usually the pectoralis minor is absent and the sternal portion of the pectoralis major, the clavicular portion being hypertrophied. It is most frequent in males and on the left side, and is no hindrance to laborious work or athletics.

The causation is obscure. Apparently it is not dependent on atavism, physical deterioration, or arrested development of nerve or vascular supply. Possibly it is due to intra-uterine compression of the upper limb against the thorax, preventing development. The side of the chest may be grooved and the hand and arm malformed. This grooving is seen sometimes in infancy and obliterated by growth in later life. Similarly, the pressure of the knee on the abdominal muscles in the right or left hypochondrium is supposed to cause atrophy and lead to ballooning of the abdomen. More probably it is a developmental defect for other deformities are often present.

*Congenital deficiency of abdominal muscles* is generally associated with genito-urinary changes, but exists without, e.g. in hernia of the cord. These rare cases are almost always male infants with no phimosis or urethral obstruction. The muscles of the anterior abdominal wall are absent or very atrophied, the lower segments being most affected. The wall may be composed only of skin. The abdomen is distended and bulged on each side. The abdominal organs are easily palpated. Distended coils, sometimes dilated ureters, are visible under the skin. The ureters are dilated and may



be convoluted. The bladder is contracted and hypertrophied ; or forms a pyriform tumour extending, when full, up to the umbilicus. The urachus may be patent. The kidneys are normal, markedly unequal in size, or fibrosed. The testes are undescended. Occasionally the external genitals are duplicated. The spinal cord shows no special characteristics. Microscopically the character of the muscle is that of non-development and not of degeneration.

The most probable explanation is a primary defective development of the abdominal muscles, which consequently render no assistance to the descent of the bladder. The ureters are therefore abnormally placed and become dilated, through partial obstruction from abnormal angle of entry into the bladder, and the changes in the kidneys are secondary. Another theory is that an abnormal blood supply produces a simultaneous embryological defect of development. A more improbable view is that the abdominal distension and atrophy are secondary to primary dilatation of the bladder.

**Myositis.**—*Simple myositis* is due to injury, microbial infection or adjacent inflammation. It may complicate rheumatism, gonorrhœa, scarlatina, typhoid, paratyphoid and other fevers. In the rectus abdominus it is most frequent as a result of fever or injury. It causes local tenderness, pain, swelling and perhaps redness, and may end in suppuration.

*Polymyositis* is primary or due to trichinæ. Occasionally it is associated with cutaneous œdema, hæmorrhage into the skin and mucous membrane, or erythema multiformæ. It gives rise to malaise, fever, anorexia, vomiting, headache and pain in the limbs. Œdema begins in the eyelids and face, and spreads rapidly all over. The muscles of the face become rigid, board-like and very painful. The other muscles become involved, especially those of the extremities, feel hard and doughy, and are very painful. The deep reflexes are abolished and there is inability to move. The course of the fever is like that of typhoid. Sometimes there is albuminuria. Some cases are subacute or chronic in course, with exacerbations. The disease is usually mild in children and gets well in a few weeks. It may last for months, or end fatally from involvement of the muscles of deglutition, respiration, and the heart. The treatment is purely symptomatic.

*Myositis fibrosa* is a rare variety of interstitial inflammation and secondary muscular atrophy. The abdominal muscles become extremely hard and the limbs contracted.

*Myositis ossificans* is a local calcification, secondary to inflammation of the muscles (e.g. rider's bone) or adjacent parts. It may be secondary to phlebitis or adenitis, the exudate into the muscle undergoing calcification. The process is analogous to that in tuberculous glands, hydatid cysts and trichinous muscle.

*Myositis ossificans progressiva* is a constitutional liability to bony deposit. It was first reported by Freke in 1740. There are now about



100 cases on record. It is not hereditary, but has been reported in father and son by Burton Fanning. It affects 5 males to 1 female. It usually begins at 2-4 years of age, sometimes not until the eighth to sixteenth year. Out of 51 collected cases (Lorenz) 11 were under 1 year, 16 between 1 and 5, and 15 between 5 and 15 years. It may be started by injury and is in this respect allied to "rider's bone."

It especially affects the muscles of the neck, shoulders, back, upper arms and the rectus abdominis; sometimes chiefly the pectorals and biceps. Later the thigh, lower arm and leg muscles are involved. Even the intercostals, masseters, temporals and platysma myoides may be affected. Occasionally it has started in the muscles of the face, but these usually escape. The muscles of the tongue, glottis, diaphragm and heart are never involved.

It sometimes begins with fever, pain, swelling and œdema of the muscles. At first the muscle is swollen, doughy and feels solid. After a variable period it undergoes ossification and bony plates, nodules or masses may be felt, sometimes intra-muscular or connected with the subjacent bone. A striking feature is microdactyly of the great toes. This was found by de Witt in 70 per cent. of recorded cases in which the condition of the toes was noted. The phalanges are short, broad and synostosed. The thumbs are often smaller than normal. In a few instances there are numerous exostoses or bony masses involving the muscles and attached to the subjacent bone.

The course is gradually progressive, sometimes with remissions in warm weather, or in successive attacks at variable intervals. It leads to progressive crippling, limitation of movement, and occasionally scoliosis and muscular atrophy. Sometimes there are long intervals of arrest. It is usually fatal in 10-12 years from pulmonary complications, if the respiratory muscles are affected, or from involvement of the muscles of mastication.

Its causation is unknown. Myositis, fibrous myositis and ossification occur successively, with the formation of disseminated nodules or plates of bone in the muscles, exostoses, and ossification of the muscular attachments. It is not congenital but may be due to an endogenous defect of the meso-blast, a constitutional state rendering the muscles abnormally sensitive to irritation. The irritation may be external, for many cases follow injury. It leads to subacute inflammatory processes, embryonal infiltration, fibrous induration and ossification. Carpenter and Edmunds examined a portion of muscle removed during life in the stage of brawny swelling and found a fibro-cellular infiltration suggestive of degeneration rather than inflammation. De Witt has described a phlebitis with an exudation, rich in osteoblasts, into the connective tissue of the muscle. Nothing can be done except protect the patient against cold and injury. Hot baths, salicylates and iodides can be tried. The disease is usually mistaken for rheumatism at the onset.



**Torticollis.**—Wry neck is due to contraction of the sterno-mastoid muscle from changes in the muscle or from tonic or clonic spasm. “False” wry neck may arise from the cicatrisation of burns, wounds or other neck injuries. In rare instances cervical caries produces a similar appearance.

An acute or simple stiff-neck follows exposure to cold, or rheumatic myositis. It is of little importance and quickly gets well when treated by local heat, massage, salicylates and alkalies. Reflex wry neck is due to reflex irritation of the spinal accessory nerve, to enlarged glands and perhaps spinal caries. The spasmodic type is practically unknown in infants, being most common in adult females and rarely a sequel of acute stiff neck. It is not limited to one muscle and is probably cortical in origin. In the paralytic variety the deformity is due to the contraction of the opposite muscle, when one is paralysed from poliomyelitis. Subsequent atrophy of the affected muscle may lead to fibrosis and contracture.

The infantile or congenital type is probably never congenital. Cases occur as a result of injury to the muscle at birth or of congenital syphilis. They are sometimes called fixed or permanent, to distinguish them from temporary, reflex, and spasmodic forms. The etiology is the same as that of sterno-mastoid tumour. Yet few cases are secondary to sterno-mastoid tumour and even gummatous deposits in the muscle do not produce it. Both factors may be absent. It is due to injury of the muscle, rupture of muscle fibres, perhaps effusion of blood, and a certain amount of myositis. This condition is rarely permanent but occasionally the muscle is replaced wholly or in part by dense cicatricial tissue. The sternal portion is the most often affected and may even be tendinous throughout, up to its attachment to the mastoid process. The trapezius, splenius and deep neck muscles may be also involved. In prolonged cases the deep fascia of the neck is shortened and the vertebral bodies are unequally compressed, becoming wedge-shaped and causing scoliosis.

The primary change is shortening of the sterno-mastoid, drawing down the head to the affected side and rotating the face and chin to the opposite side. The other changes are secondary. The face on the affected side develops imperfectly, so that there is marked asymmetry, for the carotid artery is partly compressed. If there is atrophy of the muscles and bones, the case is probably of nerve origin.

*Treatment.*—Rely on mercury, massage and manipulation, if there is congenital syphilis. In other infantile cases operation is necessary. Subcutaneous tenotomy is justifiable, if the sternal attachment alone requires division and if it stands out well above the subjacent tissues. Otherwise it is unsafe and generally ineffective, for the danger and fear of injuring underlying veins and arteries usually lead to incomplete division, and fascial bands cannot be dealt with. In the open operation an incision 1-2 ins. long is made obliquely along the anterior border of the muscle or vertically between its two attachments. The skin is retracted and a director inserted under the muscle which is divided from above. The



anterior fibres of the trapezius and fascial bands are divided if necessary. The head is put up straight or in an over-corrected position, and fixed by sandbags or a suitable apparatus. The muscle soon unites ; the child may sit up in bed in 7-10 days, or 2-3 weeks in bad cases, and get up a few days later. Recovery is complete in 3 weeks. Massage and exercises must be adopted in old standing cases or if there is any tendency to recurrence.

**Lateral Curvature or Scoliosis** is congenital or acquired. Congenital cases are rare, and due to the presence of an abnormal wedge-shaped vertebra between the eleventh dorsal and the third lumbar. This is either an extra ill-developed dorsal vertebra, with a rib attached to it on one side, or an atavistic survival.

The acquired variety is a primary lateral deviation of the spinal column and a secondary rotation of the vertebræ round the vertical axis. It is a common deformity, especially in girls of 7-14 years of age, though it may occur later in life or much earlier, even at 6 months of age. A family tendency is sometimes found. In a few instances it depends on a congenital defect of muscles and ligaments. Rickets is the chief cause in infancy and the convexity is to the left. Poliomyelitis is a potent factor in many severe cases and may occur at any age. Paralytic scoliosis usually presents only one curve, in the dorsal or dorso-lumbar region. A few cases depend on muscular dystrophies. In growing girls the chief cause is muscular weakness, the back muscles not keeping pace with the muscular development elsewhere. This may be associated with anæmia, excessive mammary development and vicious positions at work, carrying a baby exclusively on one arm or unbalanced employment of one upper limb. Many are due to faulty positions at school when writing, the curve being to the right. Others depend on inequality of the length of the legs from infantile palsy, talipes or hip disease ; persistent and severe wry neck ; or contraction of the side after empyema or pleurisy.

*Examination.*—Strip the child. Mark the spines of the vertebræ with a carbon pencil. Take note of any obliquity of the pelvis, and the level of the anterior superior spines and of the iliac bones. Suspend the child by lifting under the armpits, to see if the curve disappears. The curve may be slight, unilateral and C-shaped. Such a curve is situated in the dorsal or dorso-lumbar region, involves many vertebræ, and constantly increases from the weight of the head and upper limbs and from the muscular weakness. In the next stage the curve is double, or S-shaped, consisting of 2 equal and opposite curves in the dorsal and lumbar region. The S-shaped curve is rarely cervico-dorsal, due to torticollis or astigmatism. The primary curve is the least mobile and less easily straightened by extension. In the third stage there are 2 secondary curvatures, with lateral protuberances and flattening of the trunk. Occasionally the spinous processes project and suggest Pott's disease. In very severe and prolonged cases the rotation of the bodies of the vertebræ may cause obliteration or



reversal of the curves. The weakness of the muscles is on the convex side of the primary curve.

*Symptoms.*—The back becomes tired on standing or sitting for long. One shoulder is higher than the other, the flanks are not symmetrical and one hip projects. As the deformity increases, pain is felt on the sides of the spinous processes and is diffused in the hips, flanks or chest. Pain is due to stretching of muscles and ligaments on the convex side of the curve. Later still the deformity becomes extreme and the child dwarfed.

In the common variety the primary curve is convex to the right in the dorsal region and the secondary one convex to the left in the lumbar. The right shoulder is prominent, "is growing out," the scapula being raised and more horizontal. The right shoulder is higher than the left and the arm more closely approximated to the side. The ribs on the right side are flattened in front and project behind. In bad cases the right clavicle becomes much curved. The right flank is depressed and the hip sticks out. The ends of the spinous processes are displaced to the left in the dorsal region and to the right in the lumbar, that is toward the concave side. The transverse processes become more prominent on the convex side. The xiphoid cartilage and umbilicus are displaced to the left, and the right mamma is much less prominent than the left. The viscera are displaced. The right lung is smaller than the left, the cardiac apex is displaced upward and outward, and the liver is depressed.

The *diagnosis* is self-evident on examination of the spine. The degree of severity depends on the extent of the curve, its duration, and the amount of straightening in recumbency and on extension. It is distinguished from Pott's disease by its shape, absence of rigidity and comparatively slight pain. These two varieties of curvature may co-exist. A paralytic curve is frequently single and dorsal or dorso-lumbar.

*Prognosis.*—Under suitable treatment mild curves get well. Those due to infantile paralysis of spinal muscles and to empyemata are the most serious. Rachitic cases are bad, for the curve is liable to be accentuated during growth. In girls it is more serious than in boys, because of the weaker muscular development. The later the onset the better is the prognosis, for the parts are more fully developed. The gravity is increased, if the spine is long and flabby. The S-shaped dorso-lumbar curve is the least and the long C-shaped curve the most serious. The latter may be extreme in a couple of months. Once the condition is severe and of long standing, it is incurable, for the bodies of the vertebræ have become permanently deformed as a result of rotation and unequal pressure. If the curve does not disappear on suspension, there is bony deformity and it cannot be completely cured.

*Treatment.*—Prevention consists in teaching the child not to stand on one leg, and to sleep on a flat hard mattress with a low pillow or none at all. Excessive use of one arm and faulty positions at work must be avoided. The back of the chair should be high and sloping backward, the



feet should easily reach the floor, and the desk should be of suitable height and slope at an angle of 25-30°. Upright handwriting should be taught with the elbow on the arm of the chair, the body upright, the feet on a stool and the thighs horizontal. If one leg is shorter than the other, a suitable boot must be worn. The child should lie down in the prone or supine position for 2 hours daily, and the back muscles be massaged for half an hour. Every case of lateral curvature requires treatment, which must be persisted in as long as growth continues, in order to prevent the curve getting worse. Correction and maintenance of correction are both necessary.

Attend to the general health and for this the seaside is most suitable. Restore spinal mobility which has become impaired by contraction of ligaments and atrophy of muscles on the concave side. Teach the patient correct attitudes, and exercises planned to increase the strength of the weaker side. All exercise must be followed by a period of recumbency flat on the back. A cool or cold bath should be given in the morning and followed by rubbing with a rough towel and exercises, which bring the back muscles into play and expand the chest.

Exercises may be given at 9 a.m. and 5 p.m., followed by recumbency for 1 hour. The following exercises are suitable:—(1) Crawling on the hands and knees, with the head turned at each step toward the side on which the hand and knee are approximated; (2) Swimming movements while lying on the back or stomach; (3) Let the patient lie flat on the back on a table with the arms by the side. The arm is then flexed at the elbow and fully extended above the head by the operator, the patient resisting. (4) With the head over the edge of the table, the arms are first flexed and then fully extended laterally and above the head. (5) With the arms fully extended above the head the legs are elevated at a right angle to the body. (6) Both arms and legs are simultaneously moved in a circular manner. (7) While lying on the face, the trunk is elevated on the hands and then on the arms and the body flexed backward and forward. (8) With the legs fixed and the arms fully extended above the head the body is bent backward from the waist over the edge of the table. (9) With the patient prone the spine is manipulated by pressure on the prominent ribs to force the vertebræ more into position. At first the exercises should be easy and continued for a few minutes. They are gradually increased to more difficult ones for half an hour daily, carried out slowly with intervals of rest. Dumb-bells, not more than 1 lb. in weight, can be used in addition. Firm corsets should be worn while at lessons and after exercises.

School life is unsuitable unless the child has a special desk and long intervals for rest and games. Sculling, fencing, drilling and the use of clubs are good exercises. Supports may be necessary to relieve pain due to displacement of the viscera and to prevent, if possible, the deformity getting worse. They should only be used in incurable cases which are severe, fixed and unchanged by alteration in position, or temporarily in less



severe ones. Spinal braces are probably the best, they must not be worn continuously and must not prevent movement of the spine and muscles.

**Sprengel's Deformity**, or *Congenital Elevation of the Scapula*, was described by Eulenburg (1860) and more fully by Sprengel (1891). Up to the end of 1908 about 130 cases were on record. The scapula is in development a cervical appendage. Deformities resemble conditions found in lower animals.

The most common variety is that described by Sprengel, and is due to intra-uterine fixation of the arm behind the back or more probably to developmental error, possibly of the scapular mesoblast. It is most common in females and on the left side, but is sometimes bilateral. The scapula is elevated 2-2½ ins. above the level of its fellow and somewhat rotated, so that it is placed rather obliquely with the lower angle abnormally near the spine and fixed to it by short bands of muscle and ligaments. Elevation of the arm above a right angle is impossible and the movements of the shoulder are limited. The bone is usually smaller than its fellow and its muscles are short and defective. The neck is fuller and the neck muscles short and tense, so much so that the chin may be drawn down to the chest. There is sometimes slight curvature of the dorsal spine towards the affected side and this may lead to secondary scoliosis in the lumbar region.

In another variety the scapula is united to the spinous process of the seventh cervical or first dorsal vertebra by a triangular bony or cartilaginous ridge, the base of which is attached to some part of the vertebral border. It is probably an abnormal development of the supra-scapular epiphysis, a reversion to a lower type. It is sometimes regarded as an outgrowth from the vertebra or scapula, or an ossified rhomboid muscle. The movements are seriously inconvenienced. The arm cannot be raised above a right angle nor the hand placed to the back of the head. Congenital scoliosis is present in half the cases.

Elevation of the scapula in rare instances depends on absence of one or more muscles of the shoulder girdle, eversion of an abnormally long supraspinous portion, deformity in the upper ribs, or congenital dislocation of the humerus. Associated defects may be found in the ribs, vertebræ, humerus or clavicle; or in the adjacent muscular tissues, especially the trapezius, pectoralis major and sterno-mastoid. Sometimes there is talipes equinus, spina bifida, absent ribs or radius, and strabismus.

*Treatment.*—In simple cases exercises and massage are beneficial. The division of shortened muscles or ligaments may do a little good. The best results are obtained by the removal of abnormal bony or cartilaginous masses, when present.

**Club-foot.**—The common variety is equino-varus, an exaggeration of the normal tendency to inversion and due to malpositions in utero, spina bifida, or imperfect osseous development. It is a compound of extension and inversion. The heel is drawn up and often imperfectly developed. The foot is extended at the ankle joint, and the part anterior to the midtarsal



joint is drawn inward and rotated so that the sole is inverted. The internal border of the foot is shortened and concave, the external border convex, and there is some plantar flexion. On walking callosities develop in the skin over the external border. The upper surface of the astragalus projects on the dorsum of the foot.

The deformity can be rectified by manipulation. Even if the bones are badly shaped, persistent manual treatment is likely to be successful for they are cartilaginous and can be moulded. In untreated cases, in children who have begun to walk, the muscles, ligaments and tendons are shortened and contracted and tenotomy is needed. Later in life the bones ossify in the bad position, ankylosis takes place in some joints and new joints form.

The *prognosis* is good in infants, systematically treated. It must be guarded, if contraction has been allowed to occur, but the result is generally perfect under 15 years of age. Recurrence after treatment means that the extensor muscles have not been allowed to contract sufficiently to resist the action of gravity and cannot keep the foot at a right angle.

*Treatment.*—The varus must receive most attention. Begin treatment at birth and even do tenotomy then, if manipulation does not bring the foot into position. Manipulation is directed to overcome the extension of the foot at the ankle and the inversion of the anterior parts of the foot at the midtarsal joint. Evert and flex the foot at the ankle joint and keep it in this position for half a minute; repeat both movements for 5 minutes gently, without causing pain, 3 times daily. The anterior tibial and peroneal muscles must be systematically massaged to strengthen them and enable them to keep the foot in the right position. Continue this treatment for 2-3 months and then less often, until the child walks. Standing and walking are essential to retain the correction. If these measures are insufficient use a padded, malleable, metal splint to maintain the foot in the correct position between the manipulations. Should relapse occur on walking, a surgical boot, which will not allow inversion of the foot or extension at the ankle, is sufficient. Tenotomy is required, if the foot cannot be got into its normal position by manipulation. It is of the utmost importance to cure the inversion before dealing with the equinus and therefore to cut the tendon Achilles last. Operation, if necessary, should be done before the child commences to walk. Cases must be kept under supervision for years for relapse is common. Recovery is shown by voluntary power to correct the deformity.

**Flat-Foot.**—The ordinary or static variety of flat-foot depends on weakness of the tibialis posticus, flexor longus hallucis and peroneus longus, and secondary stretching of the ligaments which maintain the arch of the foot. It is common in rickets, debility, and as a sequel of infantile palsy. Treatment is directed to strengthening the muscles by exercises, viz., (1) Raising the body up and down on tip-toe with the toes in-turned; (2) Raising the inner border of the foot from the ground till the foot rests on the outer edge, while standing; (3) Circumduction of the foot from



without inward while sitting with the knees crossed. These movements should be repeated quickly for a few minutes, and the time gradually lengthened, and followed by a period of rest. In bad cases a metal arch may be used as an in-sole for the support of the arch of the foot while walking. The muscles must be strengthened by massage. Moderate walking exercise is advisable. Long standing must be forbidden. Preventive treatment consists in keeping fat and rachitic infants off their feet, until they are stronger. Attend to the general health.

**Enchondromata** and **Exostoses** are not infrequent in growing children. They may be hereditary or familial. Thus 4 children out of 7 were affected at the ages of 4,  $1\frac{1}{2}$  and 2 years, and at 9 months. These growths are often multiple, markedly symmetrical, and usually arise in the neighbourhood of the epiphyses. They are hard, painless, immobile, and variable in size and shape. Large ones may be more or less columnar, pedunculated, or expanded and tuberculated at the free end. Others are small, flat and sessile. They give rise to no symptoms, except such as are due to their position. They generally remain stationary and may disappear after puberty. Sometimes removal is necessary. *Enchondroma cutis* is very rare. Plates of cartilage develop in the true skin and may show signs of ossification. It has been reported in a girl 15 months old (S. Taylor and Mackenna, 1907).

**Acute Septic Diaphysitis.**—*Syn.*: *Acute Epiphysitis*, *Osteomyelitis*, *Osteitis*, *Necrosis* or *Periostitis*—*Subperiosteal Abscess*—*Acute Bone Disease*.—This is due to a trifling or severe injury and secondary infection. The injury may be a mere strain, the effect of over-exertion, or a blow or fall. It starts in or near the epiphysis, in the medulla or under the periosteum. Ill-health and local sources of infection are predisposing causes. It is a very serious disease, ending in necrosis of bone and pyæmia. It is most frequent before the epiphyses have united with the diaphyses. It may occur in the first weeks of life, is most common in the tenth and eleventh years, infrequent except at 4-12 years, and rare after the eighteenth year. The sex-incidence is 5 males to 3 females. The bones most exposed to injury are chiefly affected, especially those near the knee. Out of 165 cases the disease began in the femur 83, tibia 47, and humerus 20 (Edmund Owen).

*Symptoms.*—After a slight injury, which has apparently got well in a few days, a local tender swelling forms and the child develops constitutional symptoms, usually a rigor, and looks very ill. A local and painful swelling is found near a joint. The pain is best elicited on deep pressure. It involves the shaft of the bone, sometimes about the middle but more often near its junction with an epiphysis. Tenderness is extreme. The whole limb is often swollen and presents a white marbled appearance and dilated veins. In early stages there may be a slight red blush. The skin may be unaltered, unless there is suppuration and an abscess near the surface. All the parts surrounding the bone are thickened and give the impression that the bone



itself is enlarged. Sometimes the local signs are very slight and limited to pain, increased heat and impaired mobility. There may be no swelling, if the pus is small in amount and deeply seated. Yet the constitutional symptoms may be marked, and diminish as the local signs increase.

The symptoms are due to septic intoxication and infection. At the onset the child is fretful and cries on movement; the pulse and temperature are raised; and there is headache, anorexia, restlessness and an earthy complexion. In severe cases there are shivering, vomiting, convulsions and delirium. The child is flushed or pallid, and may sweat freely. Diarrhoea is common. Gradually the usual signs of pyæmia develop and death results from exhaustion or coma in a few days. In the most virulent type the symptoms are severe from the onset, pulse frequent and small, temperature 103-105° F., delirium early, and death may take place from toxæmia before there is much local swelling. Less severe ones end fatally from septic pneumonia, pleurisy, pericarditis or asthenia.

The whole segment of a limb may become tense, red and shiny. Remission indicates that the pus has made its way into the cavity of the shaft and tension is relieved. If it spreads under the periosteum, the tension is still high and pain and tenderness severe. It sometimes spreads to a joint by penetration through an unossified epiphysis; by tracking along the surface of the cartilage; or by intra-articular separation of the head of the bone, in the case of the hip. Hence septic arthritis is common in the hip and rare in the knee.

*Pathology.*—As a result of injury a clot forms and becomes infected by circulating germs. The most virulent and most acute cases are due to the staphylococcus aureus and the mildest ones to the staphylococcus albus. Streptococcal and pneumococcal infections vary in severity. On the diaphysis side of the epiphysis there is a layer of actively growing vascular tissue, which is continuous with a similar subperiosteal layer. Here a small abscess forms and gives rise to general infection. The effects vary according to whether it is superficial or deep. The periostitis and osteomyelitis are secondary. The periosteum is stripped up to a variable extent and the whole shaft may necrose. Sometimes the necrosis is localised at the epiphysis (acute epiphysitis) or is central. Occasionally it appears to start beneath the periosteum and ends fatally from pyæmia before pus has formed. Two such cases, affecting the humerus and femur, proved fatal in 3 days, yet nothing was found except partial detachment of the periosteum by dirty grumous fluid and pyæmic infarcts in the lungs. The chief complications are arthritis of the neighbouring joint, secondary infection of distant epiphyses, and lung complications; occasionally endocarditis and pericarditis, pleurisy, empyema, and abscesses in the cellular tissue.

*Diagnosis.*—In the first year of life the symptoms are often ascribed to gastric or dentition troubles. Later it is liable to be mistaken for rheumatism, because of the pain and swelling about the joint and perhaps septic pericarditis. Delirium with acute arthritis in one joint is not rheumatic.



The local symptoms may suggest erysipelas, but in this there is no deep-seated tenderness and the margin of the inflammation is clearly defined. The swelling of scurvy may simulate periostitis. Prolonged fever and diarrhœa, due to pyæmia, have been mistaken for typhoid fever. The main features are the unilateral distribution, the pain over the epiphysis, the thickening of adjacent parts, and the fact that the swelling is either not in or not limited to the synovial membrane.

The *prognosis* is bad. Death may result from septic intoxication, the cause of which is not even suspected. The symptoms of this subside rapidly, if the abscess is evacuated, but death may ensue from pyæmia.

*Treatment*.—Make an immediate incision down to the bone at the epiphysis. If no pus is found, explore the bone over the site of the epiphyseal cartilage. If pus is found, wash it out, drain and disinfect. In the absence of pus insert a drainage tube, dress the wound and apply a splint. Pus may appear in the wound in a short time. A timely incision may prevent pus formation and necrosis by relieving bone tension, therefore always drill the bone. The general treatment consists of liberal diet, quinine, strychnia and stimulants, and the use of suitable serum or vaccine. Subsequently necrosed bone must be removed and amputation may be necessary.

**Osteomyelitis of the Spine.**—Tubby (1905) divides this into three varieties: (1) A mild periostitic type affecting several vertebræ and generally ending in complete recovery. (2) A more severe type in which the periosteum and superficial layers of the bone are affected, causing softening and much deformity, and perhaps suppuration. (3) A virulent form in which numerous purulent foci are found in the bodies of one or more vertebræ, or necrosis of the neural arches and processes. This usually goes on to suppuration and spinal meningitis.

*Etiology*.—It is rare, most common in children and adolescents, and twice as frequent in males as females. It affects the lumbar, dorsal and cervical regions in order of frequency. It starts in the body of a vertebra as a sequel of various local infections, specific fevers or local injury. The *staphylococcus aureus* is the most common organism.

The *symptoms* are malaise and slight fever, with local pain, tenderness, stiffness and loss of mobility. The severe forms are of sudden onset with shivering or rigors, early delirium, and all the signs of severe septic intoxication. Locally there is intense tenderness, agonising pain on movement, and dilatation of superficial veins. The patient lies in a supine position. If the neural arches are involved, there is local swelling, œdema, and subsequently signs of an abscess. If the mischief is in the body of the vertebra, there is no localising sign save pain. Secondary nervous complications are pain in the limbs, hyperæsthesia, paresis, and exaggerated reflexes; paralysis of motion, micturition and defæcation; and convulsions.



Disease in the cervical region causes stiff and painful neck, inflammatory œdema of the nape, severe headache, vertigo and fits. In the dorsal region the signs are very obscure, if it is limited to the bodies, but if it affects the arches there is evidence in the back. In the lumbar region it causes flatulent distension of the abdomen.

The *diagnosis* depends on the sudden onset, rigidity of the spine, early signs of septic intoxication, and nerve symptoms. Agonising pain in the back is a most important sign. X-ray examination is useful. It must be distinguished from rheumatic and other forms of torticollis, and from Pott's disease.

The *prognosis* is favourable in the mild periosteal type. In the more severe ones 40 per cent. suppurate and two-thirds are fatal. The neural arch cases are the most easily recognised and are of better prognosis for they come under treatment earlier, but they are liable to cause spinal meningitis. The outlook depends on early recognition and evacuation of pus, prolonged recumbency, and subsequent support for the spine. In the vertebral type the pus makes its way forward or laterally as a retropharyngeal, cervical, psoas, iliac or pelvic abscess. Mild cases may be followed by deformity, whereas severe ones die before deformity develops.

**Coxa Valga** is a condition liable to be mistaken for hip disease. The normal angle of the neck with the shaft is  $110-135^{\circ}$ ; in coxa valga it is above  $140^{\circ}$ . It may be so much increased that when everted the neck and shaft are almost in a straight line. The physical signs are abduction and external rotation of the leg, limitation of adduction and internal rotation, and perhaps slightly limited flexion. The symptoms are pain, fatigue, spasm of the abductors, and limping gait with the trunk inclined to the affected side. The leg is lengthened. In standing on the affected limb the body is inclined to the same side, instead of to the opposite side as in congenital dislocation. Other signs are lumbar scoliosis, and flattening over the great trochanter which is below Nélaton's line. X-ray photographs should be taken with the feet straight, fully inverted and fully everted. Signs vary somewhat with the cause. In cases due to infantile palsy, from traction of the pendent limb, adduction may be free. The trochanter is raised, if it is associated with congenital dislocation. The treatment is purely surgical.

**Coxa Vara** and *acetabular separation of the epiphysis* have been described as separate affections, but there is strong evidence that one is a consequence of the other. Congenital cases have been reported and are probably the result of intra-uterine fracture or epiphysitis. Both affections occur chiefly among the poor, in males 14-18 years old, sometimes at an earlier age and in females. The common age of separation of the acetabular epiphysis is 1-2 years earlier than that of coxa vara. The physical signs are almost identical, varying somewhat with the duration of the injury. Possibly coxa vara is sometimes primary, due to bony softening. The angle of the neck with the shaft is under  $110^{\circ}$ , and usually a right or even an



acute angle. It is also bent antero-posteriorly, becoming convex forward.

Coxa vara is a unilateral affection, occasionally bilateral and giving rise to "scissor-leg" deformity. It may be slight or so severe that crutches are needed. The physical signs are external rotation of the hip; adduction, which may be so great that the leg is crossed over the opposite side or progression is impeded through it knocking against the sound leg; limitation of abduction and internal rotation; shortening, 1 in. or more, the upper limit of the great trochanter being above Nélaton's line.

Similar signs are produced by separation of the acetabular epiphysis, which occurs in children at any age from slight causes and gives rise to little or no pain. The leg is internally or externally rotated. In the clinical history of coxa vara we generally obtain a history of pain in the hip for a few days, of slight limping and aching due to the separation of the epiphysis, and of trauma. An X-ray photograph frequently shows irregularity in the line of the neck. The affection has nothing to do with late rickets. In view of the connection between these two diseases, it is important to examine the hip by X-rays after slight injury, and adopt surgical measures for treatment if the epiphysis is separated. Untreated cases end in coxa vara. The bone is wrenched under anæsthesia and the limb put up in an immobile position of abduction for 2-3 months. Crutches and a patten must be used for a year. In mild cases the outlook is good. In more severe ones it may be necessary to screw the epiphysis to the neck of the bone, and the prognosis is less favourable.

**Congenital Dislocation of the Hip.**—The sex-incidence is 6 females to 1 male, possibly because the capsule of the joint is more lax in the female foetus. From one-third to one-half of the cases are bilateral. It is rather more frequent on the left side. The cause is unknown. Possibly it depends on maldevelopment. It is unduly frequent in some families. Traumatism in utero or at birth is an unlikely though possible explanation of some cases. Multiple dislocations have been recorded in the newborn. It is often associated with imperfect development of the pelvis and other bones of the limb.

The character of the joint varies with the age of the child. Alterations become progressively more marked. The upper lip of the acetabulum is always defective. At first the acetabulum is circular and shallow; later it becomes triangular. The capsule is thin and slightly distended; later it becomes thicker and somewhat hour-glass in form. The ligamentum teres is thin or absent in older children. A new socket eventually forms on the dorsum ilii, adjacent to the true joint. The head of the femur is too large for the acetabulum and becomes flattened. The angle of the neck of the shaft is decreased.

The deformity may be noticed in the first month of life. Usually nothing is noted until walking begins, when a limp is observed which progressively increases. The gait is waddling, and the body inclines abruptly to the



side of the leg placed on the ground. The child generally places the heel on the ground and sways the body over the hip. Sometimes she walks on the toes. She gets easily tired and has pain in the joint when older.

Lordosis is marked in bilateral cases and less in unilateral ones. The waist is short, the buttock flattened, the leg shortened and the thigh thinner than normal. The pelvis seems enlarged, for the trochanter is very prominent and displaced backward and above Nélaton's line. The head of the bone is commonly displaced upward and backward, below and to the outer side of the anterior superior iliac spine, in front of the great trochanter, and is easily felt on rotation of the limb outward. Rarely it is subspinous or pubic in position. The displacement of the head causes an abnormal hollow in Scarpa's triangle, a concavity on the inner side of the thigh below the pubes, and asymmetry of the groove between the labium and thigh. There is slight fulness and resistance below and to the outer side of the anterior superior spine. In bilateral cases there is a distinct gap between the thighs. The two sides are rarely symmetrical. The deformity is more severe, the difficulty in walking greater, and the changes in the bones and soft parts more extensive. Passive movements are free, smooth and painless. Sometimes they are too free, on account of laxity of the ligaments, but more often they are limited in abduction and inversion. Telescopic movement, i.e. in an upward and downward direction, may be obtained.

The *diagnosis* depends on localisation of the head of the femur by X-rays or manipulation. It must be distinguished from traumatic dislocation, coxa vara, and hip disease.

*Operation* is essential, for the affection is a serious handicap because of the deformity, ungainly gait, attacks of pain, and arthritic changes in the new joint. Some children can only walk with crutches. Lorenz's bloodless method of reduction is suitable for most children under 12 years of age, especially so under 6 years. Some surgeons prefer the open operation or a combination of the two methods. There is danger of excessive damage in the bloodless method, and of sepsis also in the open method. The dislocation must be reduced and the limb put up in a position of complete abduction for 6-12 months, to allow the surrounding tissues to consolidate and prevent re-dislocation. Take X-ray photographs to ascertain if true anatomical reduction has been secured. Operation must be followed by massage and exercises for 1-2 years.

**The Painful Pronation of Childhood.**—*Syn. : Paralysie douloureuse.*—This was described by Kennedy in 1850 and by Chassaignac in 1856. It occurs in children from 1-4 years of age, occasionally older. It is most frequent in females and on the left side, for children are generally led by their left hand. The common cause is a sudden pull on the hand or forearm. The symptoms are acute pain especially on movement, complete loss of voluntary movement, pain and resistance on passive movement chiefly on supination and pronation, and the arm hangs by the side in a prone



position as if paralysed. The elbow is slightly flexed. It has been ascribed to stretching of the brachial plexus, partial luxation of the joint or epiphysis, and to psychic or inhibition palsy. Movement returns in 1-2 days and the child is well in 4-5 days.

*Pulled elbow* or *Goyraud's accident* occurs about the same age, generally in females and on the left side. It is due to strain on the forearm, and the arm at once hangs flaccid by the side with the forearm slightly flexed and pronated. Extension is impaired and supination lost. On examination there is often nothing found, but sometimes there is tenderness on pressure over the head of the radius and separation between the head of the bone and the humerus. The most probable explanation is that it is due to partial dislocation of the head of the radius. It has also been ascribed to subluxation of the interarticular cartilage of the inferior radio-ulnar articulation (Goyraud), upward displacement of the orbital ligament (J. Hutchinson, junior), compression of the interosseous membrane between the two bones of the forearm during sudden pronation, or jamming of the joint capsule at the humero-radial articulation.

It is usually regarded as a simple sprain of the elbow, wrist or shoulder, and is consequently wrongly treated. According to the description it is evidently identical with "the painful pronation of childhood." The results of treatment show that it is due to slight subluxation forward of the head of the radius.

*Treatment.*—Fix the arm, extend the elbow and sharply supinate the forearm. Then flex the elbow rapidly, while pressing with the thumb on the head of the radius. A "click" is heard and the symptoms disappear almost at once. There is stiffness, if the lesion has existed for some days, but the child can use the arm normally within 2 hours. It is liable to recur.

**Diseases of Joints.**—Primary synovitis is the result of injury or hæmophilia. Secondary arthritis is due to infective disorders, from the action of micro-organisms or their toxins, or to chemical poisons as in gout. The chief bacterial joint affections are pneumococcal, gonococcal, septic, tuberculous, and syphilitic arthritis. Less common ones are due to influenza and cerebrospinal fever. Rheumatoid arthritis, or osteo-arthritis, and pulmonary osteo-arthropathy are probably toxic. In scarlatina and other infective fevers, purpuric conditions, malaria and Maltese fever, it is doubtful whether the arthritis is bacterial or toxic. Nervous arthropathies, e.g. Charcot's joint, are rare in children.

**Pneumococcal Arthritis.**—Weichselbaum (1888) showed that the pneumococcus caused arthritis. Nattan-Larrier (1905) reported the first case in a newborn. It is the common variety of arthritis in infants. Injury to a joint is an important predisposing factor. It is a rare complication in pneumonia but may occur at any stage. Usually it is secondary to pneumococcic otitis media, to pneumococcal infection of the lungs or pleura, or part of a general pneumococcal pyæmia. Some cases are apparently



primary. Possibly in these the infection is transmitted from the conjunctiva, mouth or naso-pharynx by the blood stream.

Up to the end of the first 5 years of life purulent arthritis is commonly pneumococcal. It is most frequent in the first and less so in the second year of life. Out of 100 collected cases 31 were under 14 years of age and of these 18 were in their first and 7 were in their second year (Nitch, 1907). Out of 91 cases 28 were under 2 years and 8 between 2 and 14 years old (Herzog, 1906). It is usually monarticular and affects the knee, shoulder, hip, elbow and ankle in order of frequency. In severity it varies from a mild synovitis, with thickening and effusion of serum, to an intense purulent arthritis with erosion of cartilage and exposure of the ends of the bones. About one-sixth of the cases are serous. The contents of the joint vary from turbid serum to thick, creamy, yellowish or greenish yellow pus, and may vary in different joints, if several are attacked. The constitutional symptoms are slight unless there is severe general infection. Fever is irregular and anæmia marked. The whole limb may be swollen and œdematous. The *diagnosis* is based on the age of the patient, and the bacteriological examination of fluid obtained by aspiration. About two-thirds of the cases die. The meninges, pleura, pericardium and peritoneum may be affected. It is *treated* by incision and drainage, if the effusion is purulent. Kuster cured some cases by aspiration and injection of iodoform and glycerine.

**Gonococcal Arthritis** is rare in children. It may follow urethritis, vulvo-vaginitis or ophthalmia. Clement Lucas (1899) collected 23 cases in infants, secondary to purulent ophthalmia. The organism has been cultivated from the fluid in the joints but is only found with certainty in early stages. The arthritis may begin at any stage of the gonococcal infection, commonly in 2-4 weeks, or after apparent cure. Lucas states that it may occur as early as the fourth day or as late as the thirteenth week of infantile ophthalmia.

It is somewhat acute in onset, with effusion and no synovial thickening. Acute cases show much swelling, redness and tenderness. In less severe ones there is much effusion, with little or no redness, and less pain than in true rheumatism. Fever is slight and does not yield to salicylates. Cardiac complications are rare. When occurring in epidemic form in institutions it may cause multiple arthritis and pyæmia without evidence of a local infection. Pyæmic cases may show stomatitis, purulent infiltration of the muscles about the joint, broncho-pneumonia, empyema and high fever. The knees, wrists and ankles are very susceptible joints. The usual duration in infants is 3-5 weeks. It terminates in resolution and unimpaired mobility. Suppuration is very rare. In older children there is marked tendency to ankylosis. The *treatment* consists in curing the source of infection, protection from injury, and the use of evaporating lotions. In gonococcal pyæmia aspiration and incision may be necessary.



**Septic Arthritis** is due to direct injury or to secondary infection from epiphysitis, a suppurating wound, etc. Multiple arthritis in infants is usually pyæmic. The joint becomes acutely inflamed, swollen, hot, red and painful. The severity and fever vary. Different organisms have been found. The staphylococcus albus appears to give rise to the mildest type of inflammation. The arthritis of cerebrospinal fever is somewhat similar, but usually a mild type not requiring aspiration or incision. A few cases of septic arthritis can be cured by aspiration. As a rule incision, washing out and drainage are required.

**Tuberculous Arthritis.**—Poverty, cold, slight injuries and specific fevers are the chief predisposing causes. The knee, elbow and hip are the most often affected. The disease may start in the epiphysial end of the bone, or in the synovial membrane which becomes greatly thickened from chronic inflammation. Secondary fatty degeneration and necrosis lead to the formation of caseating abscesses. The cartilage is eroded by extension of the disease, and the bone is laid bare and becomes carious. Spongy bone is especially susceptible to tuberculosis.

It is often overlooked on account of the frequent absence of pain in the early stages. The onset is very insidious, with slight inflammation, effusion, pain, and lameness or inability to walk if the lower limb is attacked. The synovial membrane is thickened and produces the chronic "white swelling." Bone is rarefied but not enlarged. Occasionally the joint is distended with fluid and contains melon-seed bodies. Starting pains at night arise from inflamed bony surfaces rubbing together.

The course is very chronic. Adjoining muscles waste. Abscesses may form and burst, leave discharging sinuses which heal with great difficulty. It may end in complete destruction of the joint, necessitate amputation, or terminate in lardaceous disease. In early stages it is distinguished from rheumatism by its monarticular character, gradual onset and development, and the absence of cardiac complications. It is treated by rest, the usual measures adopted for tuberculosis, and fixation of the joint. The use of splints may be followed by stiff joint, but the stiffness is due to the inflammation and not to the splint.

**Tuberculosis of the Hip Joint.**—This is insidious and liable to be misinterpreted at the onset. It generally begins in the bone and may be rather advanced before it causes symptoms. The early signs are pain in the joint, limping, slight fulness in Scarpa's triangle, pain if the joint is fully extended or fully flexed, and possibly a visible focus in an X-ray photograph. In the next stage the hip is flexed and slightly abducted, due to contraction of the ilio-psoas muscle from muscular spasm, keeping the limb rigid in the position of least pain. To compensate this, there is lordosis and tilting down of the pelvis in walking. The tilting causes apparent lengthening of the limb. Comparative measurements should be made from the anterior iliac spine to the internal malleolus, with the limbs in the same position. The gluteal and thigh muscles waste. Starting pains at night tell that



the articular surfaces are involved. In a later stage there is internal rotation and adduction, due to erosion of the acetabulum and weakening of the peri-articular ligaments. The pelvis is now tilted up on the affected side and there is apparent shortening. If an abscess forms it may point on either side of the tensor fasciæ femoris, in Scarpa's triangle, in the gluteal region, or above Poupart's ligament, having made its way through the acetabulum into the pelvis and perhaps only found on rectal examination. Occasionally it invades the psoas muscle and forms a fluctuating swelling above and below Poupart's ligament.

The joint mischief is almost invariably secondary to some tuberculous focus elsewhere, commonly the mediastinal or mesenteric glands. One or more joints may be affected, most frequently the other hip. In about 5 per cent. it is bilateral. Pulmonary tuberculosis occurs in less than 1 per cent. Tuberculous adenitis is sometimes present.

The *prognosis* is good, if the disease is recognised early and properly treated. The prospect of recovery, whether complete or incomplete, depends upon the extent of the disease and efficient treatment. No case is incurable. Among the better classes bad results are uncommon. Bowlby's statistics of 900 cases at the Alexandra Hip Hospital from 1885-1908 showed a mortality of under 5 per cent. Complete recovery is possible if the disease is limited to the head of the bone, a caseous focus being seen in the X-ray photograph and the symptoms limited to slight aching and a limp. Shortening, destruction of the head of the bone, or displacement of the head of the femur on to the dorsum ilii renders a perfect cure impossible. Many cases end in severe lameness from incomplete ankylosis, displacement of the upper end of the femur on to the ilium, or arrested growth of the bone. Others end in recovery with flexion, adduction, and complete ankylosis. This is a good result, for osteotomy of the femur will straighten the limb. Speaking generally, the older the child the better is the prognosis as regards life and local recovery. Younger children are more liable to succumb from general infection, or to develop permanent lameness, for the head of the femur is small and easily destroyed.

*Treatment.*—Perseverence in local treatment is essential. The general treatment is that of tuberculosis and equally imperative. In the early stages the child should rest in the recumbent posture with the joint immobile, at the seaside if possible for at least 4-6 weeks. A firm mattress, fracture board, small pillow under the head only, shoulder straps to fix the shoulders to the bed, and a double long Liston or Thomas's splint are required. Extension is applied to the limb with a pulley and weight of  $\frac{1}{2}$  lb. per year of age up to 2 lbs., and rarely to 4 lbs. At first the extension is applied in the axis of the displaced limb, supported on an inclined plane, until the muscular spasm is overcome. In early stages this takes a few days to 2 weeks. The limb must be so flexed that lordosis is abolished and put up at this angle, resting uniformly on a pillow. Eventually it is brought down into the position of normal extension, so that the utmost value may be obtained for



it as a useful member if a stiff joint results. Extension keeps the joint surfaces apart and relieves pain. If the deformity is due to adhesion and does not yield to extension, the joint may be moved very gently under anæsthesia. Daily massage and movement of the other joints of the leg prevent stiffness.

Excision is never needed. Amputation is only required in cases of prolonged suppuration with hectic fever and destruction of the bone and joint.

The child should not be allowed to walk on the bad leg until a year after being well enough to use crutches. During this period a high boot or patten is worn on the sound limb to prevent the diseased one being brought to the ground. A Thomas's splint is only suitable for those in whom it is impossible to obtain prolonged treatment by recumbency. It is applied to immobilise the joint, as soon as the extension has overcome the spasm and the limb is straight. It must be worn for 6 months and the patient can get about on crutches. If an abscess forms it must be opened, cleared out gently, and efficiently drained. The utmost precaution must be used to prevent secondary infection with pyogenic organisms.

**Syphilitic Arthritis.**—A very rare form of symmetrical arthritis, not affecting many joints, is occasionally seen in congenital syphilis. It is a rarefying osteitis of cartilage and bone, with caseating deposit in cancellous bone. It comes on about puberty and is incurable. Possibly this is tuberculous. Syphilis usually causes sclerosis of compact bone. Occasionally a true osteo-arthritis is seen at 5-12 years of age, with much osteophytic outgrowth at the epiphyses. This is apparently a coincident rather than a syphilitic affection.

The common type begins after the second dentition, at 6-15 years of age. It is insidious in development, perhaps unilateral, but more frequently symmetrical. It almost always affects the knees and less often other large joints, especially the elbow. They are enlarged, but not tensely so, by fluid effusion and some synovial thickening. There is very little interference with function, much mobility, little or no pain, and no heat, redness, starting pains or muscular wasting. Sometimes the pain is severe. Its course is chronic and relapsing, but there is little or no destructive change. Subvarieties of this are (1) Palpable gummatous masses in the synovial membrane, with inflammation of the periosteum of the ends of the bone. (2) Osteitis and simple effusion, the end of a long bone being much enlarged. (3) Suppurative, due to secondary infection and sometimes beginning as epiphysitis in infancy. Apart from the last variety, syphilitic joint disease is associated with interstitial keratitis in 75 per cent. of the cases, and often with choroiditis or retinitis, and deafness. Both sexes are equally liable.

The diagnosis is difficult if the joint affection begins before eye symptoms. It is liable to be regarded as tuberculous or rheumatic. The child should be put to bed and a splint and Scott's dressing applied, the



latter being renewed weekly. Give mercury and pot. iod. internally. Some cases get well in 6-8 weeks; others are very intractable. If there is no definite improvement in 2 months, rely upon tonic treatment.

**Syphilitic Epiphysitis.**—This was described by Wegner in 1870 and by Parrot in 1872. It is a manifestation of congenital syphilis, and is present in most syphilitic infants which are stillborn or die shortly after birth. In others it occurs a few weeks after birth, rarely after 3 months, and only exceptionally after 6 months of life. It affects the epiphyses of one or all of the long bones. The symptoms are swelling of the limb in the region of the epiphyses and apparent paralysis, often complete immobility, of the affected limb from pain. The swelling may be limited to the epiphyses and simulate rickets. When limited to the upper end of the humerus, it is liable to be mistaken for Erb's paralysis of the upper arm type. It is often symmetrical, and may even affect all four limbs and the sternal ends of the clavicles. The epiphyses may be affected simultaneously or successively.

Inflammation commences in the layer of cartilage next to the bone. The cartilage may be separated from the bone along the line of junction, and the rubbing together of the fragments gives rise to pain, irritation and suppuration. Under proper treatment purulent arthritis is uncommon. The cavity of the joint is rarely penetrated. Secondary periostitis causes extension of thickening along the shaft and sometimes inflammation of the peri-articular tissues. Slight bone crepitation may be present, if the epiphysis is separated and not retained in apposition to the bone by the periosteum.

*Symptoms.*—When the arm is affected, the limb hangs down motionless with the hand pronated and the fingers semiflexed. Drop-wrist may be present without any local evidence of disease, if the lower radial epiphysis is alone affected. Should the lower limb be attacked, it appears to be dislocated when the child is raised by grasping him under the armpits. Voluntary movements persist in the hands and feet. The affected joint and surrounding parts are swollen, moderately hot, boggy, painful and tender. There may be very little tenderness, but usually the child cries when the affected limb is touched or moved. Pain is often so severe as to interfere with rest and sleep. The elbow, knee and wrist are the joints most frequently involved. There is no disturbance in sensibility and little or no fever. Untreated cases may eventually recover though one limb after another becomes involved. Recovery is rapid under proper treatment, and in the worst cases takes place in 1-2 months. Suppuration due to secondary infection is rare. The bone may be permanently shortened from destruction of the growing zone of cartilage. Death is sometimes due to marasmus from the underlying cause.

The *diagnosis* depends on the character of the swelling, pain, apparent paralysis, and other evidence of syphilis. Skin eruptions may appear subsequently. Rickets, scurvy and simple or rheumatic arthritis occur at a later age. Pneumococcal arthritis may give rise to difficulty. The



*treatment* consists in the vigorous use of mercury, breast-feeding, good hygienic surroundings and rest for the affected limbs. Splints are unnecessary and local applications are of little value.

**Rheumatoid Arthritis.**—*Syn.*: *Osteo-arthritis*—*Arthritis deformans*—*Rheumatic Gout*.—Although this affection has little or nothing to do with either rheumatism or arthritis in many cases, the name is a convenient one and established by long usage. Osteo-arthritis is a misnomer in that the bones are not affected in the early stages. This name should be kept for a later stage in which the cartilages, the ends of the bones, and the nervous system are affected. Possibly there are two distinct diseases. An arthritic tendency is present in some families, and may show itself in the form of rheumatic fever, rheumatoid arthritis or gout. In the disease under discussion rheumatic nodules are sometimes present, and pericardial adhesions in prolonged cases. An endocardial murmur is not uncommonly due to anæmia and dilatation, but endocarditis is rare. Acute attacks are often mistaken for rheumatic fever. The term rheumatoid arthritis is also used for the monarticular form of the disease occurring in the large joints of old people, with formation of osteophytes and the destruction of cartilage. It is a true osteo-arthritis, secondary to injury. In the disease that occurs in younger people the mischief is a peri-arthritis and the joints are little, if at all, affected. Occasional cases are seen which approximate to the true arthritic type, affecting many small joints, with the formation of osteophytes and Heberden's nodes. Thus there are two varieties, a true osteo-arthritis and a peri-arthritis. The description here given applies to the latter variety.

It may be *defined* as a specific constitutional disease, acute or chronic in its onset, of prolonged duration, liable to periods of quiescence and exacerbation, producing profound wasting in the muscles and changes round the joints, leading to crippling and deformity, but eventually coming to an end. The effects are worse than the disease. Fundamentally it is an inflammation of the synovial membrane, the bone and cartilages being usually healthy.

It is most common in girls from 15-25 years of age, and may occur in quite young children, even in the third year of life.

*Etiology.*—Nothing reliable is known about its causation. It occurs independently of soil and climate, in all classes, and in the well nourished. Due importance must be attached to a family history of arthritis and tuberculosis, to malnutrition from any cause, and to tight-lacing and to insufficient clothing. Some cases have followed infective diseases such as measles and influenza; in others there is evidence of an intestinal source, possibly intestinal toxæmia, for digestive disturbance is common. In a few patients it may be due to septic absorption from the generative tract or the respiratory passages.

*Symptoms.*—It may commence insidiously with stiffness and enlargement of the joints, or acutely and simulate rheumatic fever. Aching pains



and localised neuralgias are occasional premonitory symptoms. The fever is rarely high, 100-103° F., and is more or less continuous. It occurs in attacks of a few days to a week's duration, with intervals of apyrexia. The pulse is unduly accelerated, out of proportion to the fever, and may remain abnormally frequent after the fever has subsided. The patient presents all the signs of febrile malaise and becomes markedly anæmic. Occasionally there is albuminuria, if there is much fever. During the febrile stage the spleen and the lymphatic glands are often a little enlarged. The glands are discrete, sometimes tender, movable, rather hard, reaching the size of hazel nuts, and do not suppurate. Splenic and glandular enlargement occur in older people, but to a less marked extent. This is what one would expect, for both these structures enlarge with greater readiness in early life and are felt with greater ease.

The disease affects the small joints of the hand and to a less extent the feet. It especially attacks the metacarpo-phalangeal joints of the first and second fingers and the proximal inter-phalangeal joints. These joints present a soft, fusiform, peri-articular swelling. The joint capsules and synovial membranes are thickened; the ligaments softened and infiltrated; and the joints have a spindle-shaped appearance. Often a certain amount of effusion takes place into them. The knees, wrists and ankles are next affected, and often the disease starts in the knees or wrists. The cervical spine is affected early, and frequently the temporo-maxillary joints are attacked. X-ray examination shows that there are no osteophytes and no bony enlargement, but the bones are abnormally translucent. The amount of pain is variable and rarely as great as in rheumatic fever. In the prolonged cases the cartilages may be pitted. Adhesions of the pericardium and pleura may be found after death. Associated with the joint mischief there is pronounced muscular atrophy, as well as moderate wasting from disuse, making the deformity much more evident and the crippling more severe. The interossei and extensors are especially affected. The extremities are cold, sweating, and present a livid mottling. Freckly pigmentation appears on the limbs and sometimes brown pigmentation on the face. It may pass off as the acute symptoms subside.

*Course.*—The disease is very intractable but rarely fatal. Usually the acute attack subsides in 2-6 weeks; and the child gradually recovers from the anæmia and debility, with a certain amount of joint swelling and crippling. Recurrent, acute or subacute attacks of variable severity occur, each leaving the patient a little more deformed and a little more crippled. Finally it ceases and there remain behind the effects, viz. wasting, deformity, pigmentation, pain and frequent pulse. Occasionally mild cases are seen in which almost complete recovery takes place. If it begins in early life it interferes greatly with proper development. The true osteophytic form is rare, associated with profound cachexia and usually fatal.



*Diagnosis.*—It is most important to distinguish the disease from rheumatic fever and gonococcal arthritis, because of the treatment and prognosis. It differs from rheumatic fever in the following points. The fever does not yield to salicylates and is of longer duration; anæmia is of more gradual development; and the acid sweats of rheumatism are absent, but this is not of much assistance for they are uncommon in the rheumatism of childhood. Many joints are affected symmetrically, notably those of the hands, whereas arthritis in rheumatic fever is rarely marked in children and chiefly affects the middle sized joints. Pigmentation, local sweating of the hands and feet, and muscular atrophy are present. There is no endocarditis, but the presence of a hæmic murmur renders it difficult to exclude its existence, and in one case under my care endocarditis was undoubtedly present. An unduly rapid pulse, apart from cardiac disease, is uncommon in rheumatic fever. The possibility of the arthritis being toxic or tuberculous in origin must be remembered.

*Treatment.*—During the acute stages try the effect of salicylates or aspirin, discontinuing the drug if it does not afford relief. Colchicum and pot. iod. are sometimes useful. Wrap the joints in cotton wool and paint them twice a day with guaiacol 1 part, tr. iodi 8 parts. As soon as the fever has subsided, the patient can be liberally fed on a nutritious diet containing plentiful fat and protein. The intestinal tract must be kept in a healthy state, in view of the fact that it may be the source of the toxæmia. Subsequently reliance must be placed on warm clothing, liberal diet, dry climate, gravel soil, sunshine, open air, exercise and massage. Port, stout, ale and Burgundy are admissible. Massage may be used in the acute stages for the muscles, but not for the joints. It is beneficial to the joints as long as they are not tender or inflamed. The more the joints are used the less is the subsequent crippling. Benefit is also derived from hot fomentations, hot sand baths, mud, peat and brine baths, radiant heat, douches, massage and Spa treatment. These measures produce temporary improvement in the joint condition but do not affect the disease. For the prevention of recurrent attacks rely on general treatment, the prolonged use of cod-liver oil, and pot. iod. or the syrup of iodide of iron given alternately with arsenic for periods of 3 or 4 weeks. Good results have also followed the use of guaiacol and pot. iod. for at least 12 months. Counter-irritation of the spine by blisters, leeches, cupping or the actual cautery, in the cervical and lumbar regions of the spine, have also been recommended, on the supposition that the disease is due to lesions in the spinal cord.

**Pulmonary Osteo-arthropathy** is a rare disease in children, except in the mild form of clubbing of the fingers. It is due to suppurative diseases e.g. chronic phthisis, empyema and bronchiectasis; congenital heart disease, some liver affections and syphilis. The onset is insidious. The ends of the fingers and toes are clubbed and the nails become large, broad, brittle and



incurved. The hands, wrists, feet and ankles are enlarged. Stiffness is followed by discomfort, pain and swelling. In some the joint signs are most marked; in others the bones are chiefly affected, becoming enlarged at the ends, painful and even elongated. Effusions take place into the joints, the cartilages are enlarged and eroded, the articular ends of the bones are enlarged, and the soft tissues hypertrophied. X-rays show that increase in the size of the bone is due to symmetrical deposit of subperiosteal bone, usually on the lower ends of the tibia and fibula and the radius and ulna. It especially affects the metacarpo-phalangeal joints and metatarsals and the ends of the bones of the forearms and legs. The joint swellings may appear and disappear. The neighbouring muscles waste. It is either toxic or tuberculous in origin. In congenital heart disease it is partly mechanical, though possibly toxic substances are developed locally. Death is due to the primary or intercurrent disease.



## CHAPTER LX.

### RHEUMATIC FEVER.

Rheumatic fever is pre-eminently a disease of childhood. It is both frequent and serious. Arthritis is a symptom of minor importance, for it is neither severe nor extensive and may be entirely absent. In adults the disease especially affects the joints, whereas in children it is the heart which alone is affected or on which the main stress of the disease falls. As age advances the arthritis increases in prominence while the liability to cardiac complications, chorea, erythematous rashes, and rheumatic nodules decreases. Growing pains, epistaxis, tonsillitis, chorea and pleurisy are undoubted rheumatic signs in children.

**Etiology.**—The disease is ubiquitous, most common in temperate zones, and rare at high altitudes. It is more prevalent in some years than in others, and may assume an epidemic character for a year or so at a time. It is more urban than rural; and in London most prevalent from September to November. It is most common among the poor, the ill-fed and the badly housed. There is no reliable evidence that it is in any way connected with dampness in the air or soil, or low level of the ground water, or that it depends upon getting wet or cold.

A rheumatic family history can be obtained in about two-thirds of the cases. In childhood both sexes are equally liable. The disease is almost unknown in infancy. Only about 20 cases during the first year of life are on record, and several of these are open to criticism. Heart complications have not been noted at this early age. It is rare before the third year, infrequent till the commencement of the second dentition, and most common from that time onward to puberty. About 10 per cent. of all cases occur in the first and 30 per cent. in the next decade. It approximates to the adult type after the age of ten.

**Bacteriology.**—Klebs (1875) found micrococci in the cardiac valves. Popoff (1887) cultivated micrococci from the blood, and by intravenous injection into rabbits produced endocarditis, pericarditis and polyarthritis. Dana (1894) isolated a diplococcus from the meninges of the brain and spinal cord in a case of chorea following rheumatic fever. Many other observers have found similar micrococci. Wassermann (1899) obtained a diplococcus from chorea and produced what he regarded as typical rheumatic fever in 80 rabbits. Poynton and Payne (1900) isolated an identical organism from cases of rheumatic fever, and by intravenous injection into rabbits and



monkeys produced polyarthrititis, endocarditis, and other signs of acute rheumatism, recovering the organism from these lesions. They found it in the blood, urine, and secretion of the tonsils from the living ; in the pericardial exudate, cardiac valves, tonsils, and nodules after death. According to these observers it grows most favourably in the connective tissues, and for this reason pericarditis is severe. The most virulent cultures were obtained from the pericardial exudate. This organism is known as the diplococcus or micrococcus rheumaticus. Many competent observers have failed to find the organism during life or after death. Some regard it as a secondary infection and as a variety of salivary or fæcal streptococcus. Its morphological and staining characters are indistinguishable from those of some varieties of streptococcus pyogenes, but it does not lead to pus formation. In highly alkaline media it produces a considerable amount of acid. Ryffel and Walker found formic and acetic acids in such media, whereas 5 varieties of streptococci produced very little formic acid. They also found considerable quantities of formic acid in the urine in cases of rheumatic fever, rapidly decreasing in amount if salicylates were given. At present we cannot admit that this organism has been definitely proved the specific cause of rheumatic fever. Nor can the disease be justly regarded as an attenuated form of pyæmia. Even in acute and severe cases there is no pus formation.

**Pathology.**—There is no reliable evidence in favour of the old views that the disease is due to the direct influence of cold on joints ; to nervous lesions in the spinal cord ; or to lactic acid or uric acid in the blood. It may be mentioned that B. W. Richardson (1853) and Rauch produced endocarditis, pericarditis, and polyarthrititis in dogs by injecting a 10 per cent. solution of lactic acid into the peritoneal cavity. The theory that it is due to an infective organism, whether of one or many different varieties, is the most satisfactory explanation. Direct infection has not been proved, but cases have occurred which support the view that the disease has been acquired in this manner or by exposure to the same infecting agent. Many arguments can be advanced in favour of this hypothesis. Just as in typhoid fever seasonal waves occur. Its clinical features, viz. mode of onset, preliminary sore-throat, course of the fever and tendency to relapses, are all in favour of it being a specific febrile disease. A family predisposition exists, just as in diphtheria, scarlet fever, and enteric fever. Two children may be attacked simultaneously. Like influenza, erysipelas and diphtheria it may recur in the same patient. As in cholera and enteric fever direct infection is rare. Like malaria and syphilis there is a drug to which it is especially sensitive. Possibly infection is conveyed by dust. Both rheumatic fever and all forms of sore-throat are more common in a dusty autumn. The tonsils and naso-pharynx are probably the portals of entry for the infecting agent, although there is not necessarily a local reaction. Different observers have stated that rheumatism is preceded by tonsillitis in percentages varying from 5-80. It is certainly true that some affection of



the fauces is common, e.g. simple erythema, tonsillitis, follicular tonsillitis, and quinsy. On the other hand tonsillitis is so common that not much importance can be attached to it. It may even occur during an attack of rheumatic fever while the patient is taking salicylates.

To sum up :—All the clinical evidence is suggestive of an infectious disease ; namely, epidemic prevalence, seasonal variation, rapid onset, fairly definite course, incidence in the young, variability in type, liability to relapses, rapid anæmia, tonsillitis, heart lesions, multiple implication of joints, pleurisy, pneumonia, hyperpyrexia, nervous disturbance, liability to capillary hæmorrhages and albuminuria, erythemata and the effect of salicylates.

**Morbid Anatomy.**—The endocardium shows cell degeneration and necrosis, with secondary fibrino-cellular exudation and fibrosis. The auriculo-ventricular valves are more often and more severely affected than the semilunar, because of their blood supply and greater amount of connective tissue. The semilunar valves can only be infected through the lymphatics or by the poison in the circulating blood directly impinging on them. The myocardium shows inflammation. It is pale. The muscle fibres are large and swollen and their transverse striation almost lost. The interstitial tissue contains leucocytes and proliferating cells. Later on the muscle fibres become finely granular or fatty. Some escape and show distinct striation. The nuclei appear unaffected. The total effects are much less than in diphtheria. The pericardium may show pericarditis, patches of thickening, or adhesions, more or less universal. The synovial membrane of the joints is injected and the effusion contains some blood cells and leucocytes. Minute hæmorrhages in the heart, liver and kidneys, etc., may be present.

Subcutaneous nodules are round or oval. In size they vary from a pin's head to a pea, and occasionally that of a marble. They are single or multiple, neither painful nor tender, and better felt than seen. They are composed of collections of inflammatory lymph among fibrous tissues. They are attached to the fascia, tendon sheaths or underlying fibrous tissue about the bony prominences and joints, especially the margins of the patella, olecranon, malleolus and finger joints ; on the extensor tendons of the hands, fingers and toes ; and over the spinous processes of the vertebræ and scapulæ. Sometimes on the scalp they are extraordinarily large and numerous. They are not adherent to the skin, never suppurate, and eventually disappear and leave no ill effects. They are usually transitory, coming and going quickly ; but they may come out in crops of a few weeks' duration, or in successive crops in the same situation for several months.

**Symptoms.**—Rheumatic children are usually of a decidedly nervous temperament, emotional and excitable, and more liable than ordinary children to habit spasm, insomnia, night terrors and somnambulism. So much is this the case that the neurotic disposition is regarded as a minor sign



of rheumatism. Important minor manifestations are vague pains (growing pains) in any of the trunk or limb muscles, most frequently in the muscles of the calf or thigh. Such pain may occur at the back of the knee; may affect one hip joint and be mistaken for tuberculous hip-disease; may affect the intercostal muscles and simulate pleurisy; or may cause rheumatic torticollis. These pains may be due to a rheumatic toxæmia, for toxæmia is a common cause of myalgia.

Other signs of rheumatism are anæmia, mal-assimilation, gastralgia, epistaxis, tonsillitis, pharyngitis, pleurisy, headache, migraine, and erythematous and exudative skin eruptions. Epistaxis may precede or come on with an attack of rheumatism. Occasionally it alternates with joint pains or occurs in chorea. Rheumatic wry-neck comes on suddenly and lasts for a few days with much muscular soreness, continuous spasm, and moderate pain. It is often associated with sore-throat, and possibly the cervical joints are affected. Another feature of the disease is the frequent attacks of gastric catarrh with pain in the stomach and often severe headache. Sometimes this simulates peritonitis.

The sore-throat is a simple or follicular tonsillitis, occasionally pharyngitis or quinsy. It may disappear before the other symptoms develop. The sore-throat and flying pains may be regarded as signs of the incubation period. The disease never begins with vomiting, though frequently ushered in with digestive trouble, abdominal pain and furred tongue. The face is pale, the skin moist, but the profuse sweating, seen in adults, does not occur. The pulse is full and soft. The temperature generally rises to about 102° F., and comes down gradually in a few days unless the mischief extends. In articular rheumatism the temperature rarely goes up above 102° F., and is generally 100-101° F. Often pain alone is present, but sometimes there is well marked swelling. The joint affection may be slight or absent, monarticular, and occasionally painless. The ankles, knees, wrists, elbows, hips, and cervical vertebræ are affected in the order named. Redness is slight and often absent. There may be fugitive pains and stiffness of the joints without swelling; or the swelling only apparent on careful examination. Tenderness is limited to the joint, or involves ligaments, tendons and muscles. There is often marked muscular spasm about the affected joint. The articular affection lasts for a few hours to about 3 days.

The general condition is one of malaise with anorexia, thirst and constipation. The tongue is white and coated, but rarely presents the thick white, creamy fur seen in adults. The facial aspect is anxious, with pallor and hectic flush. Sweating is often absent, and even in children over 10 the synovitis and sweating are generally less than in later life.

The heart affection is the first and only sign in about 25 per cent. of the cases. It usually comes on in the first week, and infrequently later. It bears no relation to the severity of the attack, and is increasingly



prevalent with decrease in the age of the patients. It is present in about three-fourths of hospital cases under 10 years of age. Any portion of the heart may be affected. Endocarditis, myocarditis, and pericarditis are often insidious and unsuspected and may make rapid progress before the child is taken for medical advice or the heart affection is suspected. Some of the worst cases of heart disease are of this nature; in hospital practice a child being brought on account of marked wasting, shortness of breath, or loss of appetite. Symptoms of carditis may be absent or unnoticed for some time, or limited to mere fretfulness, anorexia and malnutrition. The pulse is always increased in frequency, though there is no positive evidence of carditis. In half the cases the heart is permanently damaged.

It is generally stated that endocarditis is the most common lesion. It is almost certain, however, that the myocardium rarely, if ever, entirely escapes. The signs of myocarditis may be limited to dilatation and increased frequency of the pulse, perhaps a murmur. Endocarditis is often unnoticed in the absence of articular pains, until serious valvular disease has developed. It may be associated with considerable pyrexia and no definite rheumatic manifestations. The early signs are irregularity and increased frequency of the pulse, slight rise in temperature, restlessness, cardiac uneasiness and a murmur. It comes on about the end of the first week and may precede joint symptoms. The murmur is almost always mitral. Its frequency is exaggerated, for in many instances it is due to dilatation and not to endocarditis.

Pericarditis is rare before the commencement of the second dentition. It is usually of the dry variety and results in extensive pericardial adhesions. It is occasionally sero-fibrinous and rarely purulent. Its onset is indicated by acceleration and perhaps irregularity of the pulse, rise in temperature, dyspnoea and restlessness. Localised friction, audible at the base and over the left auricle, may be the only evidence. It is usually associated with myocarditis (p. 482). An insidious form is not uncommon in cases of tedious and prolonged course with slight symptoms and little joint mischief; sometimes with much anæmia and headache.

Pleurisy is less frequent than in adults; multiple serositis may occur. Nephritis is rare but in one case, a girl of 7, albuminuria and casts were present for a few days. Hyperpyrexia is almost unknown. It is usually associated with pericarditis. Meyler (1908) reported a case in a boy, aged 9 years, in whom the temperature rose to 110° F., with coma, stertorous breathing and contracted pupils, hot and dry skin, violent purging and uncountable pulse. It occurred during a mild attack of the disease and ended fatally.

Anæmia is marked during and between the attacks. It may be so profound as to give rise to hæmic murmurs, impossible of diagnosis from those due to endocarditis or dilatation. The percentage of hæmoglobin falls to 60-75. There is erythropenia and a leucocytosis of 15,000-20,000.



Chorea may occur during, before, or subsequent to an attack of rheumatic fever. Its relationship to rheumatism is considered in the section on chorea. Subcutaneous fibrous nodules were first described by Barlow and Warner in 1881 as a sign of rheumatism. They may occur without any other evidence of the disease and persist for even 2 years; as in an infant, aged 17 months, whose hands were covered with nodules of this type (Carpenter, 1901). They are also noted sometimes in osteo-arthritis and I have seen them in large numbers in a girl with general tuberculosis. They are common in childhood, infrequent when youth is reached, and very rare after the age of 21 years; yet rheumatism increases in frequency and is most common from 10-20 years of age, and nearly 50 per cent. of the cases are between 20 and 40 years old. Poynter and Paine found the diplococcus rheumaticus in these nodules. If this is the true infective organism, it is strange that multiple nodules can occur without other evidence of rheumatism. Either the organism is not the causative agent, or these nodules can arise from other causes.

There is no constant or characteristic rash. Erythematous rashes are less frequent than nodules, usually accompanied with a rise of temperature, and indicate severity. The common types are an exudative erythema multiforme, gyrate or papular, *E. scarlatiniforme*, or *E. marginatum*. The patches are sharply defined, spread irregularly at the edge, fade gradually in a few days, and may cause brownish discoloration. Urticaria and various form of purpura are occasionally seen.

**Diagnosis.**—There is no distinction between acute and subacute cases, except in the degree of fever, and no indication of the ultimate result on the heart. The use of the term subacute is likely to lessen the care needed in the mildest attack. Early diagnosis is of importance because of the liability to heart mischief. Very few cases correspond to the adult type. The diagnosis depends on the association of several symptoms which apparently are independent or unrelated. The presence of arthritic symptoms is unnecessary. Make careful inquiry into the family history and a past history of growing pains, recurrent sore-throats, stiff neck, joint pain and swelling, chorea, etc. Always examine doubtful cases of fever for heart lesions and nodules. Rheumatic fever is rarely confounded with other diseases but is often overlooked. Its rarity in infancy prevents it being mistaken for scurvy, syphilitic epiphysitis and acute periostitis. Later it has to be distinguished from pneumococcal, gonococcal, septic and other forms of arthritis. The myocarditis and fever may be mistaken for early phthisis. Unilateral affection of the hip or lameness from injury may be ascribed to rheumatism or tuberculosis. Other conditions to be remembered are muscle soreness from fatigue, influenza, neuritis, and stiff neck from other causes. The most characteristic features are the migratory character of the pain, the anæmia, cardiac troubles, and the effect of salicylates.



**Course and Prognosis.**—Under efficient treatment the fever rarely lasts more than 7-10 days, and the total illness from 1-3 weeks. Even without treatment the symptoms soon subside but the heart lesions persist and progress, and on these the ultimate prognosis depends. The outlook is good as regards life, but the disease is dangerous in both its immediate and remote effects. It depends largely on the degree of myocarditis, which may sooner or later terminate in gradual cardiac failure. Pericarditis may subside in a few days or persist for 1-3 months. Although the patient may recover completely, there is a liability to progressive cardiac failure on account of universal adhesions. Even the worst attack of pericarditis may be recovered from, and present no evidence of adhesion some years later. The prognosis must be guarded in all cases of rheumatism because of the liability to recurrence and the extension of cardiac mischief with each attack. The mildest cases require treatment for 3 weeks.

**Treatment.**—Rest is of the utmost importance and must be complete, in the horizontal position, with no needless washing or movement. More especially is it necessary if the heart is affected. The ordinary methods of treatment in fever must be adopted. The child should wear a woollen night-dress, frequently changed if there is much sweating, and be covered with blankets but no sheets. A hair mattress and firm level bed are required.

The diet should consist of milk, diluted if necessary, and a liberal amount of water, barley water, lemonade and imperial drink. Farinaceous foods are added when the fever subsides. Meat extracts should be avoided, and no meat given until the child has been free from fever for a week or 3 weeks have elapsed from the onset. Alcohol is only necessary in severe heart complications.

The old systems of treatment by bleeding, purgation, alkalies, calomel and opium, blistering, and rest, have left behind some useful hints. The bowels must be kept open daily by calomel and salines. Blistering has been advocated as preventive of cardiac complications and for the treatment of prolonged joint swelling. It is unsuitable for young children, must be used very cautiously in older ones, and is of doubtful value for heart mischief. Depressant drugs, such as aconite and coal tar preparations, must be avoided. Practically no drug is used now except salicin, or some of its compounds, with or without citrate or bicarbonate of soda or potash. Stockman (1908) has shown that benzoic acid, cresotinic acid and drugs which are converted to salicylic acid in the body, namely salicin, saligenin, acetyl-salicylic acid (aspirin), methyl-salicylate, etc., have similar effects. Some drugs, practically of the same chemical composition as salicylic acid, are inert apparently because they differ in the position occupied by the hydroxyl group in the molecule. Other drugs are inert because salicylic acid is not formed from them in the tissues. Salicin, salicylic acid and salicylate of soda, preferably made from the oil of winter green and not



the artificial compound, and aspirin are the drugs chiefly used. Aspirin has the most prolonged effect for it is most slowly excreted, but it cannot be combined with an alkali. On the whole the salicylate of soda is the most satisfactory, for it can be given with an alkali and is a powerful hepatic stimulant. It should be given in full doses, with the same or double the amount of alkali, every 2-4 hours, according to the severity of the case, until the system is well under the influence of the drug and salicyluric acid is found in the urine. The dose is then reduced to 4 times a day and gradually to once a day, and continued until the patient is taking full diet. Children bear salicylates well and for long periods. The drug does not seem as efficacious as in adults, because of the small amount of joint swelling and pain. It has little influence on the other symptoms and complications, for these may develop while the child is fully under its influence. It has no marked depressant effect, but in excessive doses may give rise to giddiness, tinnitus, vomiting, air-hunger and acidosis. Poisonous effects are more apt to arise if the synthetic drug is used, if the child is constipated, and if alkalies are not given. Rheumatic fever must be regarded as a fever of 3 weeks' duration from the onset, and further treatment is essential to prevent recrudescences. If the heart is at all involved, the child should be kept in bed 5 or 6 weeks, and perhaps as many months in severe cases. Not nearly sufficient importance is attached to prolonged rest in the treatment of myocardial affections. Complete recovery from all forms of rheumatic heart disease is possible, but becomes less probable in proportion to the reduction of complete rest. The child is growing and with growth the work for the heart is increased. Therefore, it has to bear a greater strain than in adults and fresh endocarditis is easily lighted up. Complete rest from the onset takes the first place in the prevention and treatment of cardiac mischief. It should be combined with fluid diet, salicylates, diuresis and mild purgation. Counter-irritation by mustard leaf or tincture of iodine may do good in pericarditis, and does no harm in valvular disease. It is not advisable to cause actual blistering, or to apply the irritant over the cardiac area, and thus interfere with examination. Such applications can be applied just outside the limits of cardiac dulness. The treatment of pericarditis has been described (p. 498).

For severe joint pains and for the cardiac mischief, when the patient is excitable or suffers from much pain, distress and dyspnoea, a small dose of Dover's powder affords relief. The joints should be wrapped in cotton wool. Methyl salicylate and mesotan are readily absorbed by the skin. From  $\frac{1}{2}$ -1 dr. is sprinkled on cotton wool or lint, wrapped round the joint and covered with gutta percha tissue. Ichthyol is also useful. For severe pain the joints can be painted with tr. iodi., lin. chlorof. or lin. belladon.

As the patient recovers, iron is given in small doses. In conjunction with cod-liver oil and malt it affords an excellent tonic. It can be given as



iron-containing foods, in hæmoglobin compounds, or as glycono-phosphate of iron.

The preventive treatment consists of liberal diet in which there is plenty of milk and no great excess of carbohydrates. Protein food must not be diminished. Moderate hydrotherapy is good but the hardening process must be carried out with great discretion. The child should wear flannel underclothing and not be allowed to sit in damp boots or clothes. Trivial ailments should be carefully attended to for they may be rheumatic in origin and associated with endocarditis.

## CHOREA.

*Syn.: Sydenham's Chorea (1686)—Chorea Minor—Rheumatic Chorea—Cerebral Rheumatism—St. Vitus's Dance.*

Chorea was the name originally given to the dancing mania or saltatory spasm of hysteria, the true St. Vitus's Dance (*Chorea Sancti Viti*), which occurred in Germany at the beginning of the fifteenth century. Other affections of a somewhat similar name must not be confused with the disease under consideration. Among these may be mentioned electrical chorea, hysterical chorea, habit chorea, the choreic and athetoid choreiform movements which follow gross cerebral disease, and Huntingdon's chorea, a form of hereditary tremor occurring later in life.

Chorea is a bad name for this particular disease for there is no dancing or rhythmic movement, and there is much more than irregular muscular action. It is an exaggerated form of fidgets, an exaltation of the continual unrest characteristic of childhood. The movements become irregular, involuntary, disorderly, with lack of co-ordination and imperfect control.

Ziemssen defined it as "A neurosis of which the seat (as it seems) may sometimes be the brain alone, sometimes the entire nervous system, characterised by incessant twitchings or jerks of groups of muscles, which are sometimes spontaneous and sometimes excited by voluntary impulse, which occur almost exclusively in the waking state, and are accompanied by more or less developed psychical disturbance."

**Etiology.**—There is no definite heredity. A family history of organic nervous disease or epilepsy is of little importance. A neurotic temperament in one or both parents occurs in 10-20 per cent. of the cases. The most important inherited factor is rheumatism, but the figures given by different observers are so variable that it is evident that what is accepted as significant of rheumatism by one man is disregarded by others. Dana obtained a family history of rheumatism in only 8 per cent. ; Sturges found rheumatic fever in 7 per cent. of the parents, but a family history of rheumatism in



25 out of 100 cases. Goodall's figures yielded a family history of rheumatism in 17, chorea in 11, and of both in 3 per cent of 250 cases. Guthrie found it present in 47·3, doubtful in 9 and absent in 42·1 per cent. of 114 cases. H. M. Fletcher found it in an immediate relative in 25·6 per cent. of 273 out-patients.

*Sex.*—The disease is more common in females, but the younger the children the less marked is the inequality of sex. Guthrie found it a little more common in males up to the ninth year, but out of 114 cases under 14 years two-thirds were females. Out of over 2,000 collected cases the proportion of males to females was closely 1-3.

*Age.*—It has occurred in the third year of life, but cases are exceptional in the first 5 years. The most common age is 5-10, or a wider limit of 5-15, or a narrower one of 8-11. Of children under 15 more than half are in the second 5 years of life. In boys it is most common in the tenth year and decidedly rare after puberty. In girls it is rare after the nineteenth year.

Except for the fact that it is rare among negroes and Indians and common among Jews, there is no special race incidence. Nowhere in Europe is it very uncommon. It is prevalent in England, France, Germany, Austria, Italy, Russia and the United States. It is very rare in Northern India, Bombay and Abyssinia. It is more common in towns than in country districts. It is more frequent among the poor than the rich, and especially affects the weak and nervous children. It is most prevalent in winter, with a maximal incidence in December. H. M. Fletcher found it least frequent in May to September in London. A rise in the number of severe cases may be noticed in March.

Cases have followed severe fright or mental shock, generally within a week. Such an exciting cause has been found in 15·7 per cent. (Fletcher), 17 per cent. (Goodall), 22 per cent. (Koch), 25 per cent. (Gowers). Sturges found a close connection with mental shock and strain in 65 per cent. Overstudy, teasing, bullying, punishment and barometric disturbances, such as thunderstorms, appear to be exciting causes. Not much importance can be attributed to fright as a cause. The early emotional state predisposes the child to fright on slight provocation. In many instances careful inquiries will elicit evidence of the existence of the disease before the fright. Other cases have followed a fall on the head, injury to spinal nerves, or have been associated with dental irritation, roundworms, tapeworms and other local sources of intestinal irritation, with the onset of puberty, menstrual disorders, or have followed infectious fevers. Many of these affections are merely coincident.

*Rheumatism and Chorea.*—There is strong evidence that chorea is an indication or symptom of rheumatism. A family or personal history of rheumatism is often obtained. The chorea may be the first symptom, and the rheumatism may not appear for some years later. In 235 non-fatal cases Goodall found rheumatism antecedent by days or weeks in 59, by



months or years in 25. In 16 the chorea preceded the rheumatism by months or years, and in none by days or weeks. I have seen it develop in the course of acute rheumatism, and subside on the appearance of articular inflammation. A history of antecedent rheumatism has been found by various observers in percentages varying from 18-65. In 130 out of 270 collected cases, examined 2-3 years after the chorea, organic heart disease was present. Valvular disease is usually of rheumatic origin, so this is a strong argument in favour of a connection between the two diseases. In almost all fatal cases there is evidence of recent endocarditis.

Against the rheumatic theory of origin it must be noted that there are instances in which no rheumatic element can be traced during the attack, or in the family or past history. The heart is usually unaffected or, if affected, merely shows dilatation and irregularity without any evidence of inflammation. In Goodall's statistics of 250 non-fatal cases the heart was normal in 135, and 21 of these had had rheumatic fever. In 115 a bruit was present, and of these 56 had rheumatism at the time or previously. In 65 out of the 115 a systolic apical murmur was present, but no mitral regurgitation was thought to exist; of these, 20 had rheumatism at the time or previously. Rheumatic fever had occurred in 22 out of 33 with mitral regurgitation; and in 2 out of 13 with mitral stenosis. Nine had pericarditis and in 5 of these were valvular lesions. There is, therefore, considerable evidence of rheumatism, if cardiac mischief occurs. Taking into consideration the fact that a bruit is by no means proof of endocarditis, it appears that chorea, *per se*, is not a common cause of endocarditis, but that both may be symptoms of rheumatism. When chorea and rheumatism are coincident, there is no obvious relation between the gravity of the chorea and the rheumatism. Many mild cases occur though the rheumatic infection is severe.

It is a remarkable fact that nodules are not present unless there is associated rheumatism. This is very curious, if nodules are accepted as a sign of rheumatism in early life, and if chorea is regarded as a manifestation of rheumatism. Both nodules and chorea are common in childhood and rare when youth is reached. If they are both rheumatic manifestations, they ought to be frequently associated. Again, the maximal seasonal incidence of rheumatic fever is in October, showing a steady rise from July onwards, while that of chorea is much later in the year. In Philadelphia, however, it has been noted that the maximal seasonal incidence of rheumatism is only 1 month later than that of chorea.

**Pathology.**—In 1850 Germain Sée stated that "Chorea is due to the rheumatic diathesis acting on the brain and spinal cord." Many facts suggest that it is of an infective nature, and not a neurosis. The general distribution from the onset in some cases is in favour of a primary blood infection, microbial or toxic, and secondary involvement of the nervous system. The virus attacks the motor cells of the cortex and the pyramidal



tracts, the cerebral rather than the spinal parts of the nervous system. The indications of a blood affection are anæmia, diminution in the number of red cells, a pulse which is weaker and more rapid than that of health, hæmic murmurs and malnutrition. Muscular weakness may be due to the anæmia, rather than to mere lack of co-ordination.

That the poison is a microbe rather than a toxin is suggested by the frequent unilateral character of the disease. It is almost invariably more marked on one side than the other. This indicates a local cerebral lesion rather than a general cerebral toxæmia. Micro-organisms have been cultivated from the brain and pia mater, the cerebrospinal fluid, and the cardiac valves. It is probable that more than one organism can give rise to chorea, for attacks have followed many infectious diseases.

Functional instability of the nervous centres is a potent cause and may depend upon malnutrition. The disease is particularly apt to occur among the poor and the ill-nourished. It is not unreasonable to suppose that the nutrition of the cerebral tissues suffers and that an irregular, or unequal, development of the different regions ensues. On this supposition the choreic movements can be ascribed to irregular origin and conduction of impulses from the motor cells. In favour of this view it may be pointed out that many cases are cured by rest and liberal diet. The distribution and character of the movements indicate a disorder of the Rolandic area of the cortex of the brain, causing undue irritability and weakness or paresis of muscles. The psychical changes show that the whole brain is more or less affected. The spinal involvement is perhaps indicated by absent knee jerks, hyperæsthesia, pains, rigidity, weakness, muscular wasting and some kinds of ataxia. Joffroy, supported by Comby, takes a similar view and holds that, "It is a cerebrospinal neurosis of development, a disease of growth; chorea is to the nervous system what chlorosis is to the circulatory system."

At present it is advisable to regard the disease as a functional disorder due in some cases to imperfect nutrition, and in others to microbial infection which may or may not be the same as that of acute rheumatism. In many instances it is a manifestation of rheumatism.

**Morbid Anatomy.**—Extreme emaciation and excoriations of bony prominences are almost invariably present. Endocarditis is practically constant and was noted in 105 out of 115 autopsies collected by Osler. Sturges found organic heart disease in 75 out of 80 fatal cases. The mitral valve is almost always affected, and sometimes the aortic as well. Occasionally all the valves are covered by minute bead-like vegetations. They may be present in supposed non-rheumatic cases. Statistics of murmurs during life are valueless for the endocardium has been found normal, although a murmur was present. The myocardium may show patches of early fatty degeneration and foci of inflammatory exudation between the muscle fibres.



Reichardt (1902) found changes in the nervous system which he regarded as due to general toxæmia, viz. (1) Degeneration of the anterior and posterior root fibres in the cord ; (2) Small-celled infiltration throughout the brain, especially in the perivascular spaces ; (3) Small hæmorrhages, chiefly in the neighbourhood of the basal ganglia and beneath the ependymal cells of the ventricles. Neuwerck has noticed these areas of infiltration and hæmorrhage. They are not constant, and may occur in any infective disease. Poynton and Paine (1906) have found hyperæmia, thrombosis and perivascular round-celled infiltration in the brain and meninges, and chromatolysis of nerve cells. Pianese has described an ependymyелitis. Most recent observers have found no abnormality in the cerebral cortex or thalamus and no lesions in the spinal ganglia.

**Varieties.**—Different names are sometimes used according to the severity of the attack, its distribution, special characters, or the age of the patient. Maniacal chorea, or chorea insaniens, is very fatal and unknown in children. The chorea of pregnancy is associated with violent movements and fatal in about one-third of the cases. In hemiplegic chorea the movements begin on one side and are worst on, but rarely entirely limited to, that side. Sometimes there is a transference to the opposite side. Asthenic, pseudo-paralytic or paralytic chorea is a variety in which the paresis is marked and the movements slight. It may be a sequel of the sthenic or explosive variety, either mild or severe. The common form in young children is a mild chorea of long duration and moderate severity. Sometimes after chorea, clumsiness in the use of the limbs persists and the gait is blundering and ataxic. It is due to persistence of the symptoms though the disease is cured and is known as “Residual chorea.”

**Symptoms.**—The most obvious, but the least important, are the motor symptoms. The onset is insidious, except perhaps in those cases which follow severe fright. During a stage of subacute development, which lasts 1-6 weeks, the child is nervous and impressionable, laughs and cries for little or no cause, is unable to fix her attention, has a pathetic aspect, and exhibits languor, irritability of temper, trembling and awkwardness, perhaps headache and sleeplessness. She becomes anæmic, constipated, and may have variable pains. The muscles used to express emotion, and those requiring the most careful co-ordination, suffer first and most. The child is noticed to “make faces,” “drop things,” wriggle, shuffle, stumble or fall, and to be more emotional than normal. As the movements increase, a variety of emotions are expressed in the face in rapid succession and exaggerated manner, independently of feeling. Eventually the child is unable to stand, walk or sit, and even flings herself violently about the bed.

The psychical and mental state varies in character and extent. Often there is a peculiarly stupid or fatuous appearance, and the child is unduly dull, slow in understanding and answering questions, or profoundly



apathetic. This is partly the result of weakness and loss of tone in the muscles of expression, and partly from mental dulness. Frequently the child cries, smiles or laughs for little or no cause; is excitable and easily frightened; fretful and tearful, and bursts into fits of weeping. She is irritable, fearful, obstinate, with defective power of attention and impaired memory. The more severe the attack the more marked is the emotional disturbance. It may be accompanied by great excitement, pavor, hallucinations of vision, delirium and delusions. Both emotions and movements are most marked, when the child is under observation.

*Motor Disturbances.*—The muscles show involuntary movements, inco-ordination and paresis. The movements are more or less ataxic, and affect many different groups of muscles simultaneously or successively, shifting from limb to limb. They are not repeated constantly in the same groups of muscles, like the movements of habit spasm or habit chorea. They vary in severity from severe general spasms to slight twitches. In mild cases the twitches or jerks are of a fibrillary character, beginning and ending quickly, sometimes only in the trunk muscles and invisible, but felt when the hand is laid on them. Commonly clonic movements are present during rest and increased on voluntary effort. Sometimes they are continuous while at rest and disappear or are little altered on movement. In rare instances they only appear on voluntary effort. As a rule purposive movements with the extremities cannot be performed efficiently, for the motor impulse gets off the rails and stimulates unintended muscles. Spasms are first seen in the face, tongue, muscles about the mouth, zygomatics, and in the hands, especially the left hand. A good test of their presence in the hand is to make the child put the tip of the extended forefinger of each hand in turn on the end of the nose while the eyes are shut. The lack of co-ordination will be at once apparent. Even if co-ordination is brought about it is only momentarily sustained. Writing affords a fair means of dividing cases. It may be done by snatches in detached motor acts; be quite steady, there being more or less control over the movements; show much inco-ordination, although movements are absent or slight; or be quite unintelligible, even if severe movements are inhibited during the act.

The lower limbs are never affected alone and always least severely, though they may be the first involved. The movements of the legs may be so ataxic and violent that the child is unable to walk or stand. Facial spasms are never limited to one side and the trunk is always involved bilaterally. The thoracic muscles seldom escape entirely. Usually the fidgets are more marked on one side than the other, practically hemiplegic. True monoplegia does not occur, though the spasms may be most marked in one limb. The whole muscular system may be severely involved. Except in the worst cases the movements cease during sleep and recur on waking.

The decubitus partly indicates the severity. If the spasms are continual the child slips down in the bed and lies on the back to allow full play of the limbs, which stay where they fall.



The tongue is protruded with a jerk, held out by closing the teeth, and withdrawn with a still more characteristic jerk, but it is rarely bitten. Often it is large and somewhat flabby. Mastication and deglutition are spasmodic and difficult. The food reaches the mouth with difficulty, because of the inco-ordination of the hands and arms, and is bolted because the muscles concerned in swallowing and mastication are involved. Food must be soft or semi-solid. It may be necessary to feed the child by nose or rectum.

Speech defects are common in severe cases and may be the first symptom noted. They are purely muscular, due to imperfect command of respiration, to weakness of laryngeal muscles and difficulty in starting the vocal apparatus, and to weakness and imperfect co-ordination of the tongue and lips. The voice may suddenly cease or drop to a whisper in spite of violent effort, then a few more words may be jerked out, and the process is repeated till the child collapses in a flood of tears. In sthenic chorea mutism is due to spasm and inco-ordination. *Dumb chorea* occurs from paresis in the asthenic or paralytic type; speech returns as the child improves. It may be lost for weeks or months. True aphasia is said never to occur, but I cannot help thinking that in some cases the sudden and complete loss of speech is due to an effect on the speech centre.

Paresis, or paralysis, and marked loss of muscular tone are present in every case to a certain extent. It is usually monoplegic but may be hemi-, para-, or di-plegic. It may precede, coincide with, or follow the attack. The loss of power is most marked in the muscles most affected. It may be more apparent than real, being due to loss of co-ordinating power and partly to anæmia.

*Paralytic chorea*, limp chorea or chorea mollis, is most common in girls aged 7-14. The loss of power is so marked as to cause wrist-drop or an apparently complete paralysis. The whole body is affected with extreme flaccidity in 24-48 hours, or as a sequel of sthenic chorea. The choreic movements are slight and only obvious on very careful examination. Paresis has been known to be the first sign, disappearing as the spasms develop. Usually it is monoplegic or hemiplegic. A well marked case exhibits almost complete loss of voluntary movement, emaciation, prostration and imbecile expression. The lips are pursed and the upper eyelids retracted. The tongue cannot be protruded, and both speech and sphincter control are lost. Occasionally there is mania or dementia.

*Reflexes.*—The knee jerks are often normal at the onset, but later on are increased, diminished or abolished. They may disappear quite suddenly in the course of the disease, even when no drugs are given. Such loss of reflex might be attributed to arsenic in cases treated by that drug. One striking peculiarity of the reflex is the “hung-up” response to stimulation of the patella tendon. Instead of dropping immediately after the contraction the leg remains momentarily suspended, from a prolonged or choreic contraction of the rectus femoris. Or the limb gradually rises to a



position of extreme extension and rigidity and remains there for some seconds. Both the superficial and deep reflexes may be exaggerated, or one set may be exaggerated while the other is absent. The sphincters are unaffected, but the calls of nature may be neglected in severe cases. Involuntary micturition and defæcation are extremely rare.

The electrical reactions and tactile *sensibility* are generally unaltered. There may be some general blunting of sensation or mild anæsthesia, numbness, pins and needles, and occasionally hyperæsthesia. Pain in the back, joints and limbs may be due to rheumatism or to fatigue.

The external ocular muscles are rarely choreic, giving rise to momentary squint and diplopia. The *pupils* are generally dilated and frequently unequal, reacting unequally to accommodation on the two sides. True rhythmical oscillations, or hippus, are sometimes seen when the patient is quiet. At other times the variations are wide and rapid in response to light and accommodation.

The *general health* is impaired but there is no failure of nutrition, unless there is difficulty in taking food and marked insomnia. In cases of medium severity the appetite is capricious, the bowels constipated and the child anæmic. The number of red cells may be reduced to 2,000,000 per c.mm., and the hæmoglobin in proportion. Eosinophilia is often present, whereas it is absent in rheumatism. The temperature is raised, if there are rheumatic complications. Fatal hyperpyrexia has been reported by Carpenter (1907) in a child aged 3 years with post-rheumatic chorea. Respiration is often irregular, sighing, and interrupted, in consequence of deranged innervation of the diaphragm.

*Heart.*—The pulse is increased in frequency, uneven, irregular and intermittent; mainly because of the irregularity of respiration. Tachycardia is rare. Irregularity of the heart is commonly due to irregularity of breathing. It is sometimes called “chorea cordis” and is characterised by arrhythmia, a murmur, and variability in the area of dulness. If due to chorea of the cardiac muscles it persists during sleep. It is probable that there is no such thing as true chorea of the heart.

Hæmic murmurs may be present and may be ascribed to endocarditis. A pulmonary systolic murmur is often due to dilatation of the right ventricle and an apical one to dilatation of the left ventricle, or both may depend on anæmia. Dilatation of the heart is often present without causing a murmur, so the area of cardiac dulness must be carefully mapped out. It is due to anæmia, myocarditis or fatty degeneration. Pericarditis probably only occurs in those cases in which there is definite rheumatism. A slight rub is liable to be overlooked. Endocarditis gives no sign of its existence, or is indicated by rise of temperature, a systolic mitral murmur and an accentuated pulmonary second sound. The rise of temperature may be irregular and exist for some days or weeks before the murmur is heard. Rarely the endocarditis is limited to the aortic valves. It may occur



at any time in the course of chorea. A reduplicated second sound, or a definite mid-diastolic murmur at the apex, may be due to early endocarditis or commencing mitral stenosis. Reduplication at the base, frequent in children, is due to asynchronism of the semilunar valves. Nodules have been found in cases in which the heart is involved and may be present without any evidence of arthritic change.

The *urine* contains an excess of urea, uric acid and phosphates, in proportion to the movements. Urohæmatoporphyrin occurs in rheumatic subjects and was found by Garrod in 14 out of 20 cases. Albumin is rare.

*Psychical Effects.*—The temperament and disposition are generally altered. General intractability, spitefulness, and violent temper are common. After the age of 10 years these children are fretful, passionate and capricious. Dementia may occur in severe paralytic cases. Visual hallucinations may be present after 12 years of age. Attacks at puberty are sometimes associated with a state almost of terror. Mania is very rare in the sthenic stage and in children. Delirium, in acute grave cases, usually ends in convulsions, coma and death. Mental deterioration may result from relapses and moral perversion may be a sequel of long attacks.

*Complications.*—Excoriations and bed-sores will cause trouble in neglected cases and may be followed by secondary infection. Bruises, cuts, broken teeth and other injuries are due to violent movements. The most important complications are those of the heart and rheumatism. Joint effusions cause little pain and are accompanied by subsidence of the movements. Meningitis, encephalitis, and acute paralytic distension of the stomach are exceptional.

*Diagnosis.*—The movements must be discriminated from those of habit spasm, a constant source of erroneous diagnosis. Unilateral distribution suggests post-hemiplegic chorea and bilateral spasms may be mistaken for those of cerebral diplegia and other gross lesions. A prolonged duration is suspicious of organic nerve disease. The diagnosis is difficult, if there is loss of power and very little twitching. The association of hemiplegia with morbis cordis suggests embolism. In chorea the face is not paralysed, the tongue is jerky, and slight choreic movements are present in the palsied limbs. The paresis is flaccid, incomplete and of gradual onset. The severe paralytic form may be mistaken for acute anterior poliomyelitis, but some choreic movements will be noted on careful examination. Hysterical chorea and palsy are more common at a later age. In myoclonus the movements are simple and shock-like.

*Course and Prognosis.*—The mortality at all ages varies from 2-3 per cent. Death is rare in children. Mild cases tend to recover quickly and steadily in 4-10 weeks under suitable treatment by rest and food. Consequently not much importance can be attached to the drugs given. Violent symptoms rarely last more than 3-4 weeks. Bad cases may last 6 months or more. The duration is not proportionate to the initial severity. Often



the mild cases are the most prolonged. During the ordinary course of the disease there is a gradual increase in the symptoms followed by a stationary period, then a gradual decrease, and finally disappearance of the spasms from the fingers and face. Some cases exhibit an ingravescent tendency.

Severe cases terminate fatally from exhaustion and cardiac asthenia, even in the absence of endocarditis, although it is nearly always found present after death. Chorea is rarely fatal unless there is associated endocarditis and myocarditis. Hyperpyrexia, pyæmia and intercurrent disease are exceptional. A serous meningitis, such as may occur in any infection, may prove fatal. It causes a sudden and inexplicable rise of temperature, small and frequent pulse, pallor and cyanosis, and disturbed breathing. Chorea has proved fatal within 6 days of the onset. Sudden death is most liable to occur if the psychic phenomena are extreme. Dyspnœa, dysphagia, grave mental symptoms, convulsions and coma are unfavourable signs. Paralysis and loss of speech are not of serious importance. As the paralysis gets less, the movements may increase and the child seem worse, though really better. The worse the movements, the greater the inability to sleep and the more the evidence of cardiac mischief, the worse is the immediate and ultimate prognosis. Cases associated with recent articular rheumatism, a marked preponderance of heart symptoms, endocarditis and pericarditis, increasing pulse rate and respirations, are very grave. In these death is due to the heart mischief. Encephalitis is indicated by headache, hebetude, a tendency to slight unconsciousness or delirium, increasing spasms and convulsions. A scarlatiniform rash has been seen in some cases before death.

Cure is a relative term. The fibrillary movements may continue for months or years after the cessation of treatment. *Relapses* or recrudescences are frequent and often due to emotional disturbance, nightmare or thunderstorms. Relapses are more frequent in girls than boys, occurring in about a third of the cases, and may be numerous. They are more common after mild than severe attacks, are less severe than the first attack, and more likely to cause endocarditis. Sometimes they are slight cases with incomplete remissions, or really due to neurotic auto-suggestion. True second attacks generally occur after a year's interval, and the heart is more often affected than in the first attack. Muscular and mental weakness may persist for some time. Permanent injury, except cardiac, is almost unknown.

**Treatment.**—Especial attention must be directed to the heart, for here lies the great source of danger. Physical, mental and emotional rest must be insisted on. Put the child to bed for a period varying with the severity of the attack, and until free from movements for 1 week. In mild cases 3-6 weeks may be sufficient. Forcible detention in bed may be injurious, and does harm if it causes mental depression. If the child is allowed up, long hours of sleep and a rest in the middle of the day must be insisted on. The constant movements indicate an irritant lesion of the motor nervous



system. They cause tissue waste and consequent exhaustion, increased by lack of sleep. Consequently the expenditure of energy must be restricted and complete rest in bed is the best treatment. The child should see no one but her nurses, and do nothing all day. Toys, books and simple games may be allowed in mild cases. If there is mental weakness, delusions or hallucinations, isolation is still more essential. All patients recover most quickly when removed from home surroundings and the care of relatives. A gentle moral effect can be exerted beneficially by a good nurse. Mechanical restraint is injurious. Bandaging the arms to the sides, or the legs together, occasionally affords relief. The patient must not be left alone or in the dark. The bony prominences must be protected and the mattress put on the floor, with raised padded sides, if the movements are violent. Splints are rarely beneficial. The child should be amused, but not excited, helped when necessary, and the movements disregarded. Attend to all causes of reflex irritation and ill-health.

Sunshine and fresh air are advantageous. Wet packs for several hours are sedative. A hot pack for 1 hour at night is conducive to sleep. In mild and chronic cases the child may have alternate hot and cold baths each morning; a warm bath followed by a cold spinal douche; or galvanism along the spinal column.

The food must be soft, semi-solid or minced, nutritious and easily digestible. Three pints of milk may be given daily, and a liberal supply of iron-containing foods. Gavage and rectal feeding are sometimes necessary. Insufficient food is a cause of sleeplessness; a full meal with a little alcohol, given by nasal tube, is then beneficial. Use an enamel feeding cup, as china ones are apt to be broken and cause injury. Keep the bowels open.

Iron and arsenic are given to combat the anæmia. *Arsenic* is given in large doses for its supposed specific effect. Probably the apparent benefit is due to the coincidence of the passage of the sthenic into the asthenic type of the disease or to toxæmia. The large doses sometimes given are unlikely to be absorbed, for the rapid emaciation shows that assimilation is at a low ebb. Fowler's solution is given in doses of m. 10-15 t.d.s., in plenty of water, in the middle of or immediately after a meal, and given for a week, unless vomiting after the medicine comes on and indicates intolerance. The patient must be kept in bed and seen daily. A less satisfactory method is to begin with doses of m. 3, and increase by the addition of 1 drop daily until 15 or even more are taken for a dose. Arsenic may give rise to gastric irritation, foul breath, coated tongue, vomiting and diarrhœa, and dinginess, pigmentation or bronzing of the skin. It may also set up herpes zoster, erythematous and vesicular rashes, desquamation, œdema of the eyelids, albuminuria, peripheral neuritis, hyperkeratosis of the palms and soles, or even ascending paralysis. The nervous symptoms may follow comparatively small doses and come on after the omission of the drug. They may account for the loss of knee jerks and cessation of movements.



*Chloral* is a most valuable drug in severe cases and grave insomnia. Give syrup of chloral, m. 10-20, every 2-4 hours according to circumstances, and push it up to the point of inducing sleep. Large single doses are dangerous. It can be given by rectum. *Phenazone* is useful in some acute cases without cardiac or rheumatic complications. *Chloretone*, in doses of gr. 5 t.d.s. for children 7-12 years of age, is given for 2 or 3 days and then in half doses for a like period. It acts as a soporific and may give rise to skin eruptions, exfoliation of the skin of the hands and feet, loss of deep reflexes and drowsiness.

Among other drugs may be mentioned bromate of camphor gr. 3-6 t.d.s.; bromide of zinc; bromides and belladonna; belladonna in large doses, combined with potassium acetate if there is any puffiness of the eyelids or deficient secretion of urine; phenacetin and exalgin. As sedatives trional or sulphonal grs. 5 t.d.s.; hyoscin. hydrobrom. sub cutem for mania; morphine for severe restlessness, and even inhalations of chloroform. Apomorphine gr.  $\frac{1}{100}$ - $\frac{1}{50}$  sub cutem may be tried if all else fails. Ext. ergotæ liq. m. 20-60 every 3 or 4 hours, with or without liq. strychn. m. 1-2, is recommended by Eustace Smith. Trousseau also gave strychnia. D. B. Lees is an enthusiast for huge doses of salicylate of soda combined with double the quantity of an alkali. The beneficial effects may be fallacious because of the general malaise induced by the drug. He recommends sod. salicyl. grs. 20 and sod. bicarb. grs. 40 every 3 hours. To children 6-10 years old he gives 10 half-doses at intervals of 2 hours, increasing it in 2 or 3 days to three-fourths, and in another 2 or 3 days to the full dose. It is important that the bowels should be kept open. It is probable that these large doses are not absorbed, and the consequent danger to the child is reduced. The signs of poisoning are acetone in the breath and urine, air-hunger, thirst, vomiting, delirium, drowsiness, coma and death. In my experience anti-rheumatic treatment, even on this vigorous scale, is not particularly beneficial, but salicylates should be given if there is any evidence of rheumatism. In mild cases I prefer to rely on rest, liberal feeding, good hygiene and nursing, moderate doses of arsenic or arsenic and iron, and cod-liver oil and hypophosphites during convalescence. In severe ones chloral and chloretone are perhaps the most efficacious drugs.

In asthenic and paralytic cases, and acute cases with heart complications, similar treatment is adopted but stimulants and tonics are required rather than sedatives. Brandy, digitalis, caffeine, and strychnine or nuxvomica, are necessary in cardiac failure.

Massage can be used early in mild cases, with passive motions as soon as the child is sufficiently calmed. It is very useful in convalescence. At this period the improvement in co-ordination will be more rapid, if the child is encouraged in moderate use of the hands. At first she should be taught by suggestion to keep the limbs still. This may be followed by



passive movements, voluntary movements under guidance, and voluntary ones without such guidance. The usual simple games and toys, a solitaire board and bricks, etc., are all that is necessary in the way of apparatus. Later, threading beads, drawing and sewing should be practised. Supervision is necessary or the child gets careless and a state of residual chorea, due to lack of attention, remains behind. For this, drill and general supervision are required.



## CHAPTER LXI.

### TUBERCULOSIS.

*The Bacillus—Mode of Infection—Age-Incidence—Varieties—Miliary or General Tuberculosis—Tuberculous Broncho-pneumonia and Pneumonia—Chronic Phthisis.*

Tuberculosis is dependent on the activity of the tubercle bacillus in a soil rendered suitable for its growth by virtue of an inherited debility or peculiarity of structure or by modification of the tissues as the result of environment or disease. It is general or local. Its course varies from extreme acuteness and a fatal issue to marked chronicity or recovery. It is a truly acquired disease, and may be acquired *in utero*, through placental infection (p. 22). Germinal infection is incredible, in spite of the evidence afforded by Friedmann's experiments. He injected tubercle bacilli into the vaginae of guinea pigs after coitus. At the end of 1 week the bacilli were found in the foetuses though the mothers were healthy. Many cases of *Congenital* or *Fœtal Tuberculosis* are on record (40 human, 100 bovine; Huss). Transmission by this means is not very frequent. The calves of tuberculous cows do not react to tuberculin, and remain well if they are separated from the mother and brought up under healthy conditions. Many children of tuberculous parents remain healthy when separated from them. Inheritance of tuberculosis must be limited to the transmission of a suitable soil, a special proclivity. Of this there is no reliable proof, and indeed there is a certain amount of evidence in favour of inherited immunity. Probably the special proclivity is not a peculiarity but merely dependent on general debility from tuberculous as well as other causes.

The *predisposing factors* include all the causes of lowered vitality. Chief among these are lack of maternal nursing, insufficient or improper food, lack of fresh air and sunlight, unsatisfactory clothing, dirt, dust and bad hygienic conditions generally, and digestive and pulmonary troubles. Mouth and throat affections, enlarged tonsils and adenoids, and neglected teeth are liable to induce cervical adenitis and render the glands more prone to infection. Measles, pertussis, influenza and bronchitis have a similar effect on the thoracic glands. In babies and infants there is great susceptibility, just as there is to other infections. The excess of pharyngeal lymphoid tissue affords a suitable soil for the deposition and growth of the bacillus. The disease is often of slow development. The infection may be



acquired in infancy or *in utero*, to a mild extent, and may remain *latent* for years.

**The Bacillus.**—Different varieties are described as bovine, human and avian. The disease can occur in fishes. Carp in a pond were infected by tuberculous sputa and dejecta (Dubar, Bataillon and Terri, 1898). The liver and spleen were found full of tubercle bacilli. Apparently they were attenuated by cultivation in a cold-blooded creature, for inoculation into fowls and rabbits proved negative. The avian bacillus is a modified descendant from a common ancestor. It affects the abdominal organs and lungs of all kinds of birds, caged and wild; and is conveyed by infected food, inhalation and inoculation. Both the human and bovine bacillus may affect caged birds to a limited degree. The human bacillus cannot be converted into the avian bacillus by passage through birds. Of more importance is the question of identity of the human and the bovine bacilli.

Möller states that there are 9 acid-fast bacilli which stain like the tubercle bacillus. Those found in smegma, butter and timothy grass are the most important. The smegma bacillus is more rapidly decolorised by alcohol and acids than the tubercle bacillus. The best differential test is intra-peritoneal inoculation of guinea pigs. Morphology, cultural characters and virulence depend greatly on environment. The bovine bacillus may undergo metamorphosis into the human type in the human body. It is usually shorter, thicker, more irregular, and stains less uniformly than the human variety, but its characters vary with the change in culture media and are of minor importance. Culturally the bovine organism is more stabile in character, grows more slowly and with greater difficulty. According to Calmette and Guérin it grows well in the presence of glycerinated ox bile, whereas the human bacillus does not grow. Bacilli in all respects identical with the bovine bacillus can be obtained from human tuberculosis. Those of human type can be converted into the bovine type by "passage" experiments. They are therefore grades of the same species, growing less freely on human soil. The bovine bacillus is the more virulent one and produces general tuberculosis in guinea pigs, rabbits, pigs, goats, bovines and apes. The virulence of the human bacillus can be increased by passage through calves, rabbits and cats. Bovine tuberculosis can be produced in various animals by inoculation with the human organism, and there is no distinguishing features between the tuberculosis produced by these organisms (Report of the Royal Commission, 1904). Many of the contradictory results of animal inoculation with the human bacillus have been due to insufficient dosage, for undoubtedly it is much less virulent than the bovine organism. It is merely a question of dosage and susceptibility of the particular animals used for experiment. The Royal Commission found that the human bacillus produced general tuberculosis in guinea pigs, monkeys and anthropoid apes; and limited lesions in oxen, rabbits, pigs and goats. The bovine bacillus, even if of human origin, caused generalised



tuberculosis in bovines and monkeys. The bovine organism is more frequent in children than in adults, and is said by some observers to be almost limited to children under 10 years of age. It chiefly affects the abdomen and glands. In this connection it is interesting that animals are practically immune from the adult type of phthisis, and that adults rarely have caseous mesenteric glands unless there is intestinal ulceration. Children often have such glands, and are less liable to the adult type of phthisis. It is curious, if true, that the virulent bovine organism is apt to cause generalised infection or a localised glandular one, and that the milder human bacillus is more likely to set up pulmonary tuberculosis of the chronic excavating type.

**Mode of Infection.**—The disease may be introduced by the placental circulation; inoculation of the skin, mucous membranes or subcutaneous tissues; the alimentary or the respiratory tract. Contagion in the home is common in infancy and early childhood. The infection is conveyed by kissing, coughing, infected handkerchiefs, etc., from a phthisical parent or nurse; by creeping on the floor and absorbing infected dust; through the food supply, or even from a midwife. Reich (1878) reported a remarkable instance. Two midwives divided between them the obstetric practice of a town of 1300 people. Both practised mouth to mouth aspiration and blowing into the nose of the newborn. One was tuberculous and within 14 months 10 of her infants died from tuberculous meningitis. Those under the other midwife were unaffected. Infected dust is conveyed by the fingers to the mouth or nose, especially in the second year of life during the crawling stage. Dieudonné of Würzburg (1903) examined the dirt on the hands and the nasal secretion of children, aged 9 months to  $3\frac{1}{2}$  years, and found tubercle bacilli almost always present. The results were confirmed by inoculation of guinea pigs. Numerous experiments have shown that in coughing and loud speaking droplets of spray containing tubercle bacilli are disseminated. It is obvious that the liability to infection is great, if a child of susceptible age is brought in contact with a case of open tuberculosis, and that the utmost precautions must be adopted to prevent this mode of infection.

Accidental infection of a wound by sputa and tuberculous inoculation of a circumcision wound are rare occurrences. The two great channels of infection are the alimentary tract from the lips to the anus, inclusive of the tonsils and pharynx, and the respiratory tract. The impossibility of separating these two systems in the mouth and naso-pharynx has led to great difficulty in ascertaining whether infection is alimentary or respiratory in origin. Tubercle bacilli in air inspired through the mouth may be deposited on the mucous membrane and cause local infection, enter the lymphatics or blood stream without causing a local lesion, or be swallowed. Admission of the occurrence of intestinal infection does not logically lead to the assumption that the infection is of bovine and not of human origin.



The frequent introduction of the bovine organism into the human body may have an immunising effect or may perpetuate the disease. The matter is of great importance in view of the opinion held by many that infected milk is the chief cause of tuberculosis in children.

*Sociological Considerations.*—If milk infection is the main cause, the disease should be most prevalent among children brought up on cow's milk and least among the breast-fed. Kitasato states that the cattle in Japan do not suffer from tuberculosis. The infants are suckled for 2-3 years and cow's milk is little used for infants. Yet tuberculosis, including the intestinal type, is as common in children and adults as in other countries. In India infants are suckled 2-3 years. Tuberculosis is rare in cattle, and all milk is boiled and butter converted into "ghi" before use. Glandular, peritoneal and pulmonary tuberculosis are rare. Infants are suckled 2-3 years in Egypt, and both glandular and peritoneal tuberculosis are rare. In China artificial feeding of infants is unknown, yet tuberculosis is very prevalent. In Bohemia Biedert found that the incidence of phthisis varied directly as the density of population, although the urban dairies were under better control. Heller found intestinal tuberculosis more prevalent round Kiel than in Berlin where milk is boiled or pasteurised. These observations, except as regards Japan and China, are not of great assistance. They suffice to show that milk infection is not always the cause and suggest that it is not the chief cause. Tuberculosis is more common among the poor than the better classes, although they use less milk. It may be that they obtain inferior and contaminated milk and that it is given uncooked. On the other hand the possibilities of direct infection are much greater among the poor; and there are many cases of infantile tuberculosis in all classes in which the child has never taken cow's milk in any form. In 9 out of 17 successive hospital patients under 1 year old there was strong reason to suppose that infection was direct.

*Milk Infection.*—Stress has been laid on the frequency of tuberculosis in cattle and of bacilli in the milk. It is stated that 2 per cent. of all milk cows in England have tuberculous udders and that 50 per cent. show some tuberculous lesion in the carcase. Klein found tubercle bacilli in 7 out of 86 samples of London milk examined in the years 1904-1906. There is no doubt that cows are liable to disease of the udder, that tubercle bacilli are not infrequent in milk, and that they may be present although there is no udder affection, even in large numbers. The bacilli may be numerous in the fæces of affected cows.

According to the Report of the Royal Commission (1907) tuberculosis, especially in children, is in certain cases the direct result of infection by the bovine bacillus and in most of them the infection is carried by the milk. Cow's milk containing the bovine bacillus is clearly a cause of tuberculosis. A very large proportion of tuberculosis contracted by ingestion is due to the bovine bacillus. Tuberculous cow's milk ought never to be used as food.



These conclusions were based on feeding experiments which showed the bacillus highly pathogenic to animals in the following descending order :— (1) Chimpanzee, baboon, rhesus monkey, lemur, rabbit and guinea pig ; (2) Pig, goat, calf, cat and dog, the adults being less susceptible than the young ; (3) Rats and fowls apparently insusceptible. A remarkably small dose was needed for anthropoid apes. The infection may be localised in the mucosa, may leave no local trace and pass into the glands, or may quickly reach the lungs although there is no trace in the mucosa or glands. The bronchial glands are almost invariably affected after the lungs, sometimes alone.

Von Behring maintains that the disease in adults is due to the recrudescence of infection, latent since infancy, and that it is the result of the ingestion of tuberculous milk in infancy. Caseous, calcified and healed fibrous foci are frequently found after death from all causes, according to Naegeli in 97 per cent. If the resistance is lowered from any cause, a latent tuberculous process may become active and disseminated. But there are other sources of infection as well as milk.

*Feeding experiments* must be carried out with great care, in order to exclude the possibility of infection via the tonsils and pharynx. Calmette and Guérin (1905) inoculated the udders of goats through the milk ducts with human, bovine, avian and timothy grass bacilli. Those inoculated with the last two organisms did not react subsequently to tuberculin, nor did their kids develop tuberculosis. The bovine bacillus proved fatal in less than 2 months, though there was little extension beyond the udder. Their kids showed extensive infiltration of the mesenteric glands. Those inoculated with the human bacillus, and their kids, were less severely affected.

Infective material was introduced by tube into the rumen of goats and kids. The bovine bacillus set up acute dissemination in kids in the mesenteric glands, lungs and mediastinal glands; in one, the lesions were limited to the mesenteric glands. In the adult goats extensive pulmonary mischief was induced, with little affection of the mesenteric glands. This suggests that pulmonary phthisis in the adult is of intestinal origin and that the glands have less protective power than in earlier life. Similar experiments by Whitla and Symmers of Belfast (1908) on anthracosis, by the introduction of carbon particles into the intestine, showed that the lungs were more readily infected in adult animals and that in the young the mesenteric glands acted as a barrier. These glands should therefore be often infected in children.

These and other experiments show that the tracheo-bronchial glands and lungs can be infected by ingested tubercle bacilli, without any infection of the intestinal tract. It must be noted however that the pulmonary lesions are miliary in type, a general tuberculosis, and not like those of chronic phthisis.



A *local lesion* is not essential. In feeding experiments the cervical glands of the pig become tuberculous although there is no lesion of the mucous membrane. The mesenteric glands of guinea pigs and other animals become infected apart from local mischief. Tubercle bacilli have been found in tonsils with an unimpaired mucosa. Sputum rubbed on the nasal mucosa of animals produces tuberculosis but no local lesion. It is a curious fact that intestinal ulceration shows little tendency to dissemination and may be very extensive without any caseation of the mesenteric glands. The ulcers are probably due to infection carried by the blood or lymph. The small tuberculous ulcers found in the stomach are almost undoubtedly due to this mode of infection and not to infection by ingested bacilli. These glands frequently contain virulent bacilli. MacFadyen and MacConky (1903) examined the glands of 20 non-tuberculous and 8 tuberculous children, 26 under 2 years of age. The mesenteric glands yielded virulent bacilli in 5 tuberculous and 5 non-tuberculous cases, though in 7 of the 10 microscopic examination of the glands was negative. They also found the organism in the glands of a still-born child.

We must conclude that the bacillus can pass through the stomach uninjured, probably more readily in infants because of the deficiency of HCl. The intestines show no local lesion. The mesenteric glands may appear healthy under the microscope yet may contain virulent bacilli. The organisms reach the thoracic duct, the vena cava superior, the right side of the heart, and are distributed to the lungs. Oberwarth and Rabinowitch (1908), by inserting bovine bacilli through a gastric fistula with the œsophagus closed in sucking pigs, brought forward evidence that the bacilli pass into the blood, lungs and other viscera in 22 hours. Such a result may be possible in infants. If the organisms are not detained in the lungs, they will pass to the left side of the heart and may reach the meninges, setting up meningitis, and thus account for those cases in which no tuberculous focus is found.

The bacilli can be detected in the chyle of dogs within 3-4 hours of ingestion. Nicholas and Dercas found the chyle, collected from the thoracic ducts of guinea pigs fed on an emulsion of bacilli, was fatal to dogs. Ravenal fed dogs on the bacilli through a stomach pump and found the organisms subsequently in the chyle and glands. Similarly the bacilli have been found in the lung capillaries after the ingestion of tuberculous food. From the evidence it may be justly concluded that the ingestion of tubercle bacilli is a cause of tuberculosis in the thorax.

Tuberculosis has decreased greatly in the last 50 years, except in the early years of life. This is somewhat in favour of milk infection. Although the infectivity of such milk must be admitted, it may not be so great as commonly supposed, for there are numerous other modes of infection. Alimentary infection is not necessarily from milk. It is quite likely that many children receive a mild bovine infection and are thereby protected or



rendered immune. Mild infections must be very common, for the specific organism is widely distributed. It is impossible to state definite distinctive features between a tuberculosis of bovine and one of human origin. The bovine bacillus is more virulent for all animals and perhaps for the child. The number of bacilli which get into the system must be taken into account. The large number, due to contact with a dirty phthisical patient in unhygienic surroundings, do much more damage than a few scattered throughout a milk supply. The milk may be contaminated by human tubercle bacilli.

*Air-Infection.*—The bacillus is conveyed by spray, dust or direct contact. One c.c. of sputum may contain a million bacilli (Bollinger) and a single patient may expectorate 4000 million in 24 hours (Nuttall). Fortunately many are dead, but it is obvious that the risk of infection from contact with a case of open phthisis is considerable, especially among the poor and unclean. The inspired bacilli are deposited on the mucosa of the naso-pharynx, mouth, tonsils, larynx, trachea or large bronchi. There is no certain proof of direct infection of the lungs by inspired air. Inhalation of the bacilli into the alveoli is most improbable, if not impossible. Vallée found calves very little susceptible to direct infection of the trachea or naso-pharynx. The inhalation of carbon particles does not apparently cause anthracosis, if the œsophagus be tied. On the other hand inhalation experiments on animals usually cause lesions of the lungs and bronchial nodes only; possibly through ingested bacilli. Against the inhalation theory it may be pointed out that, although infants and young children are extremely susceptible, primary tuberculosis of the larynx or bronchi is almost unknown. Air-infection probably means that the bacillus enters through the mucous membrane of the throat or alimentary tract. Direct infection of the lungs is negligible.

*Tonsillar Infection.*—The tonsils, adenoid tissue of the pharynx, and cervical glands are frequently affected. Possibly in chronic phthisis they are coincident and not the primary source of infection. The frequency of tuberculosis of the tonsils has been considered (p. 237.) Cervical adenitis does not prove infection through the mucous membrane of the mouth or pharynx. Calmette, Guérin and Breton infected the deep cervical glands of guinea pigs by the introduction of bacilli into the stomach. The whole lymphatic system, as well as the lungs, can be thus infected. Grober claims to have demonstrated a direct route from the tonsils, through the cervical lymph nodes, to the lungs and pleura. A. Most made careful injection experiments with coloured liquids and seems to have proved that there is no anastomosis between the cervical and subclavian lymphatics and those of the pleura and mediastinum; and none between the lymphatics of the mesentery, liver and diaphragm and those of the pleura and mediastinum. Tuberculous infection from cervical glands takes place via the thoracic duct and blood stream.



**Post mortem Evidence.**—Only a limited amount of importance can be attached to the apparent age of a tuberculous focus, as indicative of the primary source of infection. It may have completely healed and the disease be due to further infection. Localisation of the lesion affords little evidence of the portal of entry. There may be no local lesion and the focus situated in some distant region from the point of entry of the bacillus. Buhl stated 100 years ago that there is no phthisis without an antecedent caseous focus. Certainly it is extremely rare not to find such a focus, if the autopsy is done thoroughly. In children there is a remarkable tendency to infection of the lymph nodes. After the third year cervical adenitis may be the first or the only sign of infection. The thoracic glands are peculiarly liable, especially those on the right side and the pre-tracheal gland. Although these glands may be large and caseous, it is possible that general tuberculosis is due to further alimentary infection. For the symptoms of tuberculosis of thoracic and mediastinal glands, *vide* p. 520-523. Different observers have found the thoracic glands tuberculous in 73-100 per cent. and the mesenteric ones affected in 35-78 per cent. The disease is much more often limited to the thorax than the abdomen.

It is clearly established that the thoracic glands are often alone involved and much more frequently than the mesenteric. Possibly the former are more susceptible or more perfect filters. Bollinger injected tuberculous sputum into the peritoneal cavity and found that the peritoneum remained sound in two-thirds, though the lungs became tuberculous. And much experimental evidence shows that these glands can be infected by organisms carried from the intestines, without evidence of disease in the intestinal mucosa or the mesenteric glands. In most cases there is an isolated caseous area in the lungs and the corresponding gland is the one affected. The disease is primarily pulmonary, though the local lesion may be healed, healing, or even undiscoverable. Possibly in a few instances the bacillus has been conveyed to the glands without causing local mischief in the lungs. The cervical glands are affected in 80-100 per cent. of fatal cases.

The *lungs* are constantly affected to some extent. Froebelius gives the order of frequency of the pathological changes in infants, aged 1-4 months, as :—lungs, bronchial glands, liver, spleen, intestines, meninges, kidneys, mesenteric glands, heart and pericardium, pleura and upper air passages. Occasionally the tonsils, pharynx, middle ear, skin, bones and joints are attacked. The lungs were infected in 78 per cent. of 269 cases under 12 years (Still) ; in 96 per cent. of 54 deaths under 13 years (Pendlebury Hospital) ; in all of 72 children under 2 years (Holt). These figures are enough to show the extreme frequency of lung infection. Cavities may be of considerable size, even under 6 months of age. In none of 933 fatal cases did Baginsky find the lungs alone involved.

**Primary Intestinal Tuberculosis.**—Accepting as primary intestinal tuberculosis only those cases in which the lesion was limited to the intestinal



mucosa or the mesenteric glands, with no trace in the lungs or bronchial glands, Albrecht only found 7 in 1060 autopsies on tuberculous children under 12 years. Other observers include cases in which the lesions are extensive and of old standing, with fresh foci elsewhere ; and those in which the disease is so far advanced, in comparison with that in other foci present, as to be probably of an earlier date. But the site of the oldest lesion is not always the source of recent infection. Nor is a more advanced focus necessarily an earlier one. Consequently statistics based on these data are not of much value as evidence of primary intestinal infection. In England they are given as 18-25, Berlin 1·5-25, France 2·5, and America 3 per cent. Councilman of Boston regarded 37 per cent. of tuberculous cases as abdominal. Certainly primary intestinal ulceration is rare. Raw found only 3 cases in 600 autopsies at all ages, and all under 3 years of age. Intestinal ulceration is not necessarily bovine. It may be absent though the lungs are diseased and much sputum is swallowed. Snow reported a case in a 3-months old baby, the infection having been acquired from a consumptive nurse. Apparently abdominal tuberculosis is much more frequent in Glasgow and Edinburgh than it is in English or Continental cities.

*General infection* is usually the result of ulceration of pulmonary vessels by a caseous focus, possibly in the adventitia, or through the thoracic duct. A more localised infection may arise from the breaking down of a small focus and ulceration into the lumen of an artery. The tuberculous toxin destroys the cellular and interstitial tissue, and modifies the growth of the cells, producing spindle and giant cells. Finally the newly formed cells undergo cheesy degeneration.

**Age-Incidence.**—The disease is much more frequent in early infancy than commonly supposed. Out of a series of 127 autopsies at the Belgrave Hospital for Children, on infants under 1 year old, 17 were tuberculous, 5 of them under 6 months. Of 54 cases of tuberculous meningitis under 5 years old, 10 were under 1 year. Froebeliu found that 4 per cent. of 18,569 autopsies, on infants dying between 1 and 4 months of age in the St. Petersburg Infant Asylum, were tuberculous and that in all the lungs were involved. Post mortem evidence shows its existence in 2-6 per cent. of infants under 3 months ; 10-15 per cent. in the first year ; and 25-40 per cent. under 15 years of age. These are by no means constant figures, being sometimes considerably lower, e.g. 11 per cent. under 15 years (Orth), sometimes higher, e.g. 58 per cent. (Fibiger). Ghon found no healed lesion under 3 years of age. Of 110 cases 60 per cent. died from tuberculous meningitis, and in 64 out of 67 it was secondary to tuberculosis of some other organ ; 23 died from miliary tuberculosis ; 27 showed lesions such as are seen in adults ; none were primarily intestinal and in only 4 were the mesenteric glands affected.

The mortality statistics of the presence of tuberculous foci show that the disease increases in frequency but diminishes in severity as age advances.



Both autopsies and tests during life prove that it is less frequent during the first 2 years than later. The mortality is greatest during the first 2 years, being highest in the second year, because of the danger of home infection and the liability to general tuberculosis and meningitis. In conjunction with the fact that the disease is common in countries in which cow's milk is not tasted, this is an argument against milk infection. But in those places in which cow's milk is used the frequency of fatal tuberculosis in the first 4 years of life, the age of milk feeding, is an argument in favour of milk infection. After 2 years of age tuberculosis accounts for about half the deaths in children, but the mortality from all causes is small at this period of life.

Percentage Age—Incidence of Tuberculosis fatal under 4 years.

	No. of cases.	1st.	2nd.	3rd.	4th.
Brandenberg ..	203	18	42	21	19
Kingsford ..	243	29	37	22	12

Of Kingsford's 324 cases (*Lancet*, 1904) under 10 years old, 162 died in the first 2 years and 270 in the first 5 years of life. Freeman of New York (1904) stated that out of 158 cases 61 died in the first year and 34 in the second. These figures are sufficient to show the general incidence of the disease. The mortality is lowest from 5-20 years of age.

Tuberculous foci are found with increasing frequency as age advances. The increase is rapid up to the end of the first year, gradual up to the end of the fifth year, and slow afterwards. In the sixth year such foci are present in about half of the autopsies; from 10-15 years in about 75 per cent. Statistics, based on the clinical examination of school children, are unreliable for the signs are often indefinite and may be misinterpreted. Probably from 1-2 per cent. are affected among those actually attending school. Those seriously ill are kept at home.

**Varieties of Tuberculosis.**—The tendency to generalisation in the form of miliary tuberculosis or of multiple tuberculous foci is a characteristic of early life. The other main peculiarity is the liability to glandular disease. Except in glands, and not often in these, it is rarely localised in a single organ. Usually there is an aggregation of yellow tubercles at the oldest focus, and miliary or yellow tubercles scattered elsewhere. The disease may preponderate in the thorax, abdomen, cervical lymph nodes, joints or other sites. In the first 4 years of life the bronchial glands are almost invariably affected and the lungs little or not at all involved. As already argued it is probable that there is, or has been, a primary focus in the lungs and the glandular infection is secondary. At the time of death, however, the lungs may show signs of secondary dissemination or spreading by continuity with a broken down gland. Often the glands are much affected, while the lungs show merely a few miliary tubercles. General tuberculosis



may be due to dissemination from foci in other glands, tonsils, adenoids, etc. In rare instances no such focus is discoverable and it is possible that the disease is due to direct infection by tuberculous milk or dust. At this early period of life there is marked proclivity to bronchitic and pulmonary affections which render the tissues more prone to infection, and to marasmus from numerous causes reducing the vitality of the child and its resistance to infection.

Chronic bronchitis is often due to tuberculous disease. Sub-acute caseous broncho-pneumonia is not infrequent. The middle zone of the lung is the most often affected, and the lower lobe next in frequency. Occasionally caseous nodules, some of which have broken down and formed small cavities, are profusely distributed throughout the lungs.

The chronic phthisis of adults is rare in the first 4 years. After the seventh year pulmonary tuberculosis closely conforms to this type. It is more diffused, more malignant, showing less tendency to heal and greater tendency to dissemination, and rarely follows a regular chronic course. All these features decrease in prominence as age advances. According to Louis's Law, after puberty the lungs are almost invariably affected when any other organ suffers.

*Differences from Adult Tuberculosis.*—Tubercles develop rapidly and coalesce, caseation is almost constant, and fibrosis and calcification are rare, especially before the fifth year. The disease often starts from the glands at the root of the lungs. Such a gland may soften and ulcerate into the trachea or bronchus, setting up diffuse caseous broncho-pneumonia. Often it spreads by the lymphatics along the bronchial septa; and small cavities at the root of the lung, or in 1 lobe, or radiation outward toward the periphery may be present. The meninges, brain, glands, bones and joints are involved more frequently.

No matter the mode of infection the disease is disseminated rapidly and irregularly through the lungs, and often simulates broncho-pneumonia. Typical tubercles and giant cells are numerous. Cavities are quite common, though less so than in adults. Death usually results from dissemination before the cavities are large enough to give rise to typical physical signs. On account of the course of the disease the physical signs are different. Thus in general caseation, without cavitation or large areas of consolidation, the signs may be merely those of bronchitis. The phthisical appearance of adults is rarely seen in children under 10 years of age, who may look pretty and healthy until removal of the clothes reveals emaciation.

*Early symptoms* are debility, loss of weight, liability to colds, morning anorexia, headache, sweating, fever and variable crepitations. Night sweats are uncommon. Such sweating comes out on the forehead and round the neck. It is inconstant and worse in cold weather. Heavy bed clothes and rickets must be excluded as causes. Hæmoptysis and laryngitis are rare. Sputum is swallowed and may set up gastro-intestinal troubles,



so it is advisable to give calomel and intestinal antiseptics. The skin is often dry, discoloured and pigmented. Fever may be absent, atypical, or due to mixed infections. The tuberculosis of glands and other forms of localised disease are described under the special headings. In the present chapter general dissemination and the various lung affections are considered.

*The Blood.*—Leucocytosis is a sign of secondary infection. An increase in eosinophiles is a favourable, and decrease is an unfavourable indication.

**Miliary or General Tuberculosis.**—This is the common type of tuberculosis of the lungs in infants. From 2-5 years it is much less frequent. After that it is uncommon but may occur at any age. It was present in 48·3 per cent. of 238 children (Still), and in 73 per cent. of 110 (Hamburger and Sluka). It is due to blood infection. The irruption of the bacilli into the blood stream is the first scene in the last act of infantile tuberculosis. Possibly more than one such irruption can take place. Its cause is the breaking down of a caseous gland, usually a thoracic gland, and infection of the lymphatics; or the breaking down of a nodule in the wall of a blood vessel. In the latter case dissemination may, if arterial, be limited to a definite area in the lung, abdomen, meninges, etc.; if venous, the bacilli are carried to the heart, thence to the lungs where they are mainly detained, while those which escape may be distributed by the arterial system generally, no organ being exempt. The focus may not be discoverable. In older children the disease may be secondary to tuberculosis of bones, joints, glands, testicles, etc.

Miliary tuberculosis is almost invariably a general tuberculosis. The tubercles are distributed throughout the lung, more or less equally, or in patchy areas. They are often in enormous numbers and of the small grey type, passing into yellow tubercles in prolonged cases. The lungs may be otherwise healthy, exhibit general catarrh, or show patchy broncho-pneumonia. The visceral and parietal pleura are often studded with grey tubercles, sometimes the under surface of the diaphragm, and less often the peritoneum. A variable number are found in other organs, chiefly the spleen.

The *onset* is insidious or abrupt. Wasting, without definite cause, may be present on account of the tuberculosis in the glands. Often the child appears in robust health and the onset is that of capillary bronchitis or broncho-pneumonia. Anæmia, anorexia, malaise and fever may be present, before the physical signs appear, and there may be large numbers of tubercles in the lungs without signs of pulmonary mischief.

*General Symptoms.*—The child is ill, weak, feverish, and frequently emaciated. The weakness and emaciation are progressive. The skin is dry and in marasmic infants may develop small furuncles. Blueness or lividity of the lips and face may be noted; increased respiration rate; cough frequent, dry, sometimes paroxysmal, often slight or absent in infants; frequent pulse, unless there is meningitis; and often a diazo-reaction in the urine. The temperature is irregular, rarely above 102° F., seldom



hectic. It should be taken every 2 hours in doubtful cases. Fever may be trivial or absent.

*Localising signs* are secondary, and often absent or late in development. The chest resonance is somewhat diminished. Fine râles and occasionally friction may be heard. The liver and spleen are generally enlarged, and the latter may increase in size because of the fever. The liver is often fatty. Further evidence of dissemination may be present, e.g. meningitic symptoms, choroid tubercles, glandular tuberculosis, epididymitis, etc.

The disease assumes *various types*. (1) Marasmic :—seen in infants and characterised by wasting, diarrhoea, exhaustion, little or no fever, and perhaps no pulmonary signs; (2) Meningeal :—the common type in which the meningitic symptoms mask the lung signs; (3) Pulmonary :—insidious or acute in onset and simulating general broncho-pneumonia; (4) Acute Febrile :—suggestive of enteric fever and characterised by high continuous or remittent fever, frequent pulse, wasting, prostration and enlarged spleen. The dyspnoea and cyanosis are greater than the pulmonary signs warrant. The pulse is more frequent, fever more irregular than in enteric fever, and there is no leucopenia, no Widal reaction and no spots. Exceptionally spots, closely resembling rose spots, have been seen.

*Diagnosis*.—The marasmic type is often impossible of diagnosis. If there is other obvious cause for fever and wasting, do not be in a hurry to diagnose tuberculosis. In infancy debility, marasmus and congenital syphilis lead to wasting which may be associated with pulmonary catarrh, a condition liable to be mistaken for tuberculosis. In older children the wasting of intestinal dyspepsia, with cough and some fever, may lead to similar error. Acute cases may simulate capillary bronchitis and broncho-pneumonia in infancy and enteric fever at all ages, occasionally sepsis, infective endocarditis and malaria. From capillary bronchitis it cannot be clearly differentiated. Much respiratory distress, in the absence of physical signs, is strongly in favour of general tuberculosis. Many a case which gets well is diagnosed as capillary bronchitis or broncho-pneumonia; and many with similar symptoms and physical signs prove to be miliary tuberculosis. Choroid tubercles, bacilli in the cerebrospinal fluid, and the various tuberculin tests are sometimes of assistance. After the second year of life these cases are usually diagnosed as meningitis, because of the predominance of meningeal symptoms. Unhygienic surroundings, pre-tuberculous conditions, and association with phthisical subjects must be allowed due weight.

*Prognosis*.—The disease is fatal in 3-6 weeks from the onset of acute symptoms, occasionally earlier. Possibly mild outbreaks of miliary tubercles into the lung are recovered from, just as similar outbreaks in the peritoneum get well. One child under my care recovered from an attack which appeared to be a capillary bronchitis. About 6 months later a like attack ran a similar course and ended fatally, and miliary tuberculosis was found.



There seems no inherent reason why a patient should not recover from the irruption of a crowd of bacilli into an artery and distributed locally.

**Tuberculous Broncho-pneumonia.**—This variety is common in infancy, and broncho-pneumonic or pneumonic in distribution. It is more prolonged in its course than general tuberculosis. It is stated to be sometimes a sequel of simple broncho-pneumonia, especially one due to measles or pertussis. Localised cases can depend on direct spread from a caseous gland to the lung. Generalised ones are most likely a sequence of rupture of a caseous focus into the trachea or bronchus and aspiration of the débris. This may affect a large portion of a lobe, a whole lobe or 1 lung, and is rarely bilateral. It is usually basal and causes cheesy consolidation, tending to softening and cavitation. Section of the lobe may show miliary tubercles extending from the primary focus in a radiating or fan-shaped manner into the lung. Apart from this variety tuberculous broncho-pneumonia is more frequent at the apex than the base. The pleura is generally thickened and adherent.

*Symptoms.*—Acute cases are like simple broncho-pneumonia in onset, course and physical signs. Others resemble lobar pneumonia but there is no crisis. Bilateral cases are analogous to acute phthisis or galloping consumption, with yellow tubercles about the size of peas, some softened, throughout the lungs and partial consolidation round each. The physical signs of this type are those of catarrh, and impaired resonance if there is much consolidation. As a rule the disease is more insidious and runs a prolonged course, with abatements and exacerbations, and even intervals of comparative good health. The common symptoms are pallor, languor, anorexia, wasting, dyspnœa, cyanosis, and variable cough which may be absent at first. The physical signs are dulness, bronchial breathing and fine crepitations; and subsequently large moist râles and perhaps signs of a cavity. The most reliable indications of a cavity are loud bronchial breathing, altered voice resonance, pectoriloquy, splashing and bell sound. Cracked-pot sound is of little value in infants and not much in older children. On account of the yielding chest wall it can be produced in health. In chronic cases the signs may be limited to 1 lobe, with moderate fever, malaise and cough. Hæmoptysis seldom occurs, except when a broken down gland extends into the lung and invades the bronchus or trachea. Then the bleeding may come from the lung or from softening and rupture of an artery in the gland. Sometimes the disease is quiescent, the signs of consolidation alone being present.

*Diagnosis.*—Simple broncho-pneumonia is distinguished by its acute onset, absence of factors disposing to tuberculous infection, and absence of the signs suggestive of tuberculosis. Both varieties may last for months, but the longer the course the greater is the probability of tuberculosis. Pleuritic attacks and downy hair on the trunk, common in chronic tuberculosis, are in its favour. It may be possible to obtain sputa and discover the bacillus, or the organism may be found in the little masses of purulent



matter in the stools when the sputa are swallowed. The lobar distribution may simulate unresolved pneumonia. Basal cavitation is also found in bronchiectasis. The temperature chart may suggest typhoid fever and a complication.

*Prognosis.*—Acute cases end fatally in 6 days to 6 weeks, while chronic ones last 6 weeks to 6 months. Malignant cases are often fatal in 2 weeks; galloping phthisis in 10-12 weeks; and others pass into chronic phthisis. Those occurring in the course of measles and pertussis are very unfavourable; to a less extent so, if the primary infection has subsided. The most favourable cases are those of limited area. Though quiescent there is always a liability to sudden and fatal dissemination from trivial illness. Fibrosis is a rare termination and on the whole acute dissemination is not common. Death may take place during the acute stage from asthenia or convulsions, due to general tuberculosis or meningitis.

**Tuberculous Pneumonia.**—At all periods of childhood tuberculosis of the lung may be of an acute pneumonic type. The whole of 1 lobe or 1 lung undergoes massive caseous consolidation, which may terminate in extreme caseous disintegration.

It is more or less abrupt in onset, with chill, high fever and signs of consolidation, and may resemble simple pneumonia. In a few days the fever becomes hectic. On exploration pus may be obtained, if the needle enters breaking down lung, a caseous pulmonary abscess. The fever continues, dyspnoea increases and cardiac weakness ensues, ending fatally in a few days. Or it passes into galloping phthisis with hectic temperature, rapid wasting, asthenia, caseation and death in a few weeks. It may be mistaken for empyema.

**Chronic Phthisis.**—The adult type occurs at all ages, but is rare until the fifth year and more frequent after the seventh. There is less tendency for it to begin at the apex and extend gradually downward. The base of the lung is more often involved than in adults but there is nevertheless a preponderance of apical cases, increasing as age advances. Physical signs are often found over the root of the lung, about the mammary region, because of the spread of the disease from a bronchial gland. These glands are usually much affected and cause pressure symptoms. There is less tendency to fibrosis and to extensive cavitation. Cavities are found at all ages, even in the first 3 months of life. They are frequently situated in the apex of the lower lobe, the anterior and inferior half of the upper lobe, and the root of the lung. The liability to general dissemination and meningitis is much greater than in adults.

In infants and young children the *physical signs* are those of bronchopneumonia, for the pathological process is of this character. Such signs, of gradual onset and limited to one apex, are probably tuberculous. Older children present similar signs to those in adults, viz.—deficient movement, unilateral flattening, impaired note; breath sounds weak and harsh, jerky,



tubular, bronchial or cavernous; expiration prolonged and perhaps louder than inspiration; râles, sometimes absent in early stages, and later of varying size, clicking, bubbling, etc. Cavernous breathing, pectoriloquy and cracked-pot sound may be present over consolidation as well as cavitation. Impaired resonance over the apices of the lower lobes in the intra-scapular region may be due to the rhomboid muscles. Sometimes there is extremely little found on examination, and the physical signs are limited to a unilateral or general increased resistance on percussion, a less resonant percussion note than expected, weak breath sounds and a few fine crepitations at the edges of the lungs. The signs of enlarged thoracic glands may be present.

The *symptoms* consist of fever, cough, dyspnœa, hæmoptysis, wasting, and increase in the pulse rate and respiration. The pulse is almost invariably increased in frequency. Dyspnœa is not marked but breathing is usually somewhat more frequent than normal. Cough is slight or absent, dry and irritating, bronchitic or paroxysmal. It is less common than in adults. Sputum is swallowed, except by older children who have been taught to expectorate, and may cause vomiting if it is purulent and abundant. Hæmoptysis is very rare under 3 years of age and uncommon later, occurring in about 5 per cent. It is rarely fatal in children under 7 years. It is due to congestion, rupture of a blood vessel, or perforation of the trachea or bronchus by a necrotic gland. Fever is common, without special features. It may be continuous in an acute case, perhaps not above 100° F., or may have a daily range of 3-4° F. Sometimes it keeps quite steadily about the normal level. Emaciation is almost constant. A few of the older children, less often the younger ones, remain plump, robust and of a good colour, and feel well. Night sweats are uncommon in early life. The older the child, the more likely are they to occur.

*Diagnosis.*—Prolonged broncho-pneumonia is liable to be mistaken for phthisis. If the signs and symptoms can be thus explained, it is rash to diagnose tuberculosis. Stress must be laid on exposure to infection, localisation of the pulmonary signs, asymmetry of respiration above and below the clavicle, loss of weight, and the temperature chart.

*Complications* are common. Infection of the blood stream causes dissemination, meningitis and even endocarditis. The presence of endocarditis in a girl with high fever and pulmonary catarrh rendered the diagnosis between general tuberculosis and infective endocarditis a matter of difficulty for some days. In chronic types there may be secondary infection of the tonsils, larynx or intestines, softening of caseous glands and its effects, and abdominal tuberculosis. In chronic phthisis every variety of tuberculous lesion may be found, as well as those due to other organisms or dependent on malnutrition.

The *prognosis* of the adult type is better than that of other varieties but must be guarded on account of the limited tendency to fibrosis, the



liability to dissemination, and the danger of further outbreaks in apparently cured cases. Such an outbreak may be due to fresh infection. Quiet and extinct lesions have been found post mortem in 9 per cent. of 2,400 autopsies (Kingston Fowler, Middlesex Hospital), 71 out of 763 (Loomis of New York), and 17 out of 131 in the Paris Morgue (Vibert). It runs a prolonged course and recovery is not infrequent, even in infancy. The fact that it is of this type implies resistance to general infection or lack of virulence of the infective agent. As a standard rule it must be remembered that the prospect of recovery increases with the increased age of the child affected and is slight in the very young. The severity seems to be exaggerated at puberty in girls, especially if the catamenia have begun. Pressure signs are of grave significance. Enlarged bronchial glands without pressure signs are less serious although there is a prospect of secondary general infection.

**Specific Tests for Tuberculosis.**—Seek for bacilli by *staining methods* in the sputum, fæces and cerebrospinal fluid. Sputum is obtained by irritating the pharynx with a gauze swab and collecting it as it is coughed up, by getting the child to cough on to a glass slide, by washing out the stomach in the morning before food, or by aspiration through a catheter introduced into the larynx.

The withdrawal of 1-2 c.c. of blood and *inoculation* of guinea pigs is rarely available and takes at least 3 weeks for the effect to be ascertained. The estimation of the *opsonic index* (p. 91) is not very reliable. *X-ray examination* may afford evidence of old lesions or of enlarged thoracic glands. *Cyto-diagnosis* (p. 434) is of value in effusions.

The *cutaneous reaction* of von Pirquet is simple and free from danger. Inoculation of 25 per cent. old tuberculin into the scratched skin causes the formation of a flat red papule within 24 hours. A simple and reliable method is to make parallel and crossed double scratches on the forearm with two sewing needles held together and dipped in undiluted tuberculin. A control scratch must be made as well. Or scrape off the epidermis without causing oozing, and rub in the tuberculin. Comby makes 3 pricks with a lancet in the deltoid region and rubs in 1 per cent. tuberculin. In 24 hours or more redness appears and is followed by simple papules which last 8-10 days. The papules may be vesicular, bullous, and rarely show a central eschar. The skin reaction is indicative of the presence of a tuberculous focus but affords no information as to its latency or activity. It is valuable as a negative test in infants and young children. It is positive in about 90 per cent. of tuberculous and 20 per cent. of non-tuberculous children.

The *ophthalmic or conjunctival reaction* was described by Wolff-Eisner in 1907 and by Calmette in the same year. Use a fresh 0.5 per cent. solution of old tuberculin in normal saline or 3 per cent. boric acid. If there is tuberculous disease it sets up conjunctivitis in 3-12 hours. The reaction is supposed to depend on the circulation of tuberculous toxin in the tissues.



It is a deceptive and unreliable sign and may do great damage. It has set up violent conjunctivitis, chronic conjunctivitis, a local tuberculous process and ulceration of the cornea, and even total destruction of the eye. The solution recommended here is unlikely to cause a violent reaction. Advanced cases may not show the reaction. The test is negative in 10-15 per cent.

The *tuberculin test* consists in the injection of old tuberculin m.gm.  $\frac{1}{10}$ - $\frac{1}{2}$  and recording the temperature at short intervals. It rises slowly in 4-6 hours, more rapidly in another 2-4 hours, and falls to normal within 24 hours. It may remain up more than the 24 hours or the rise may be delayed. If there is no reaction, the test can be repeated with doses of m.gm. 1-5 at intervals of 2 days. It must not be employed if the axillary temperature is above normal, or in the presence of hæmoptysis, bacilli in the sputum, definite physical signs of tubercle, grave cardiac or renal disease. The sole risk is recrudescence of activity in a tuberculous focus. A local lesion shows swelling, redness and tenderness.

**Treatment.**—The *prophylaxis* of tuberculosis is so well known that it may be summed up briefly. The avoidance of exposure to infection and increasing the resisting power of the body are the two chief factors. Therefore, keep children away from tuberculous relatives or nurses. Bring babies up on the breast or select a milk supply from non-tuberculous cows. Take special care of them during and after respiratory catarrh, lung affections, pertussis and measles. Attend to hygiene. Open air by night and day, sea air, inland air, moderate clothing and judicious hardening; liberal diet, containing plentiful fat and protein; breathing exercises, drill, physical development, games and sports; and the conditions of school life, are all of great importance. Definite cases should not attend school and, if active, should be segregated in the home.

For *active cases* adopt the methods of prophylaxis and open air principles of treatment, viz.—a sparsely populated locality near the sea or a pine forest and free from dust, dry soil, equable temperature with no wide extremes, bracing and not relaxing climate, shelter from east and north winds, and a large airy bedroom. Seaside air is best for pre-tuberculous stages and glandular tuberculosis, and inland air for pulmonary disease. High altitudes are unsuitable for children. Over-fatigue and exposure to any infection, even that of the common “cold,” must be guarded against. Give a cold or tepid morning bath and dry with a rough towel.

*Tuberculin Treatment.*—Koch’s old tuberculin depends for its activity on the extra-cellular toxins excreted into the culture fluid. It is rarely used for treatment and, if so, is given in doses 0·001 m.gm., is eventually increased to 1 m.gm. for adults. Begin with 0·0001 m.gm. for an infant, and 0·00025 m.gm. for a 4-year old child. Malaise, fever, increased pulse rate and loss of weight indicate too large a dose. Koch’s “New” Tuberculin T. R. is generally given, according to the method already described (p. 92). The signs of improvement are better appetite, colour, general health and



gain in weight, and local healing. One c.c. of "new" tuberculin is standardised to represent 10 m.gm. of dried comminuted bacilli. The active agent is an intra-cellular toxin. Tuberculin can be given by mouth in double the doses recommended for subcutaneous injection. Apparently it is almost as efficacious when given in this manner. Its value is difficult to estimate, for the cases which improve are those that usually do well under ordinary methods of treatment.

The injection of tuberculin, hetero-inoculation, is unsafe in large doses and if the patient is feverish. Human tuberculin is preferable to that of bovine origin, but the latter may be used if there is no improvement. Auto-inoculation causes a rise of temperature and a high temperature means over-dosage. This is reduced by rest. If the temperature falls the prospect is favourable. The course of auto-inoculation is shewn best by a chart in which a series of evening temperatures are joined together (E. C. Hort). A rise in the curve is unfavourable, while a fall is a good sign. In surgical and chronic tuberculosis in which auto-inoculation does not cause rise of temperature, supplementary hetero-inoculation may prove of decided benefit.

*Marmorek's serum* is given per rectum in doses for adults of 5 c.c. daily or 10 c.c. twice a week. The opsonic index rises after a few doses, soon reaches a maximum and remains there for 3-4 weeks, while the serum is given, and does not fall until nearly a week after its omission. Its value, like that of other serums and vaccines, is uncertain.

Apart from these methods the treatment is *symptomatic*. Education need not be neglected, if the child is over 6 years old and the disease not acute, and can be given in the open air. Massage is often useful and stimulants, preferably malt liquors, may be needed. The diet should consist of small frequent meals, much milk, protein and fat. Raw meat is taken well. Usually too much carbohydrate is given. Children do not stand much over-feeding. Prescribe rest before meals.

*Drugs.*—Be careful not to upset the digestion. Give ol. morrhue dr.  $\frac{1}{2}$ -1, t.d.s., after meals, except on Sundays. Give it in the form most agreeable to the child, e.g. with malt and hypophosphites. Other drugs of value are ichthyol in capsules, ichthalbin in chocolate tablets, creosote m.  $\frac{1}{2}$ -1 (p. 80), guaiacol carbonate gr. 1-5, terebene, arsenic, arsenicated waters, and preparations of iron (p. 81).

Cough depends on irritation of the fauces, pharynx, larynx or trachea. It is treated by local demulcents; a simple linctus or one containing tr. camph. co.; liquorice, glycerine, cocaine or morphia lozenges; honey and lemon juice; ammon. chloride spray if there is much tenacious mucus; bromides at night and heroin, etc. Inhalations of terebene, eucalyptus, tr. benzoin co., creosote, etc., are also beneficial. D. B. Lees is a warm advocate for the continuous inhalation of a mixture of ac. carbol. drs. 2,



creosoti drs. 2, tr. iodi. dr. 1, sp. etheris dr. 1, sp. vin. rect. drs. 2, about 5 drops being put on a respiratory hourly and continuously inhaled.

Hæmoptysis is treated by rest, morphia gr.  $\frac{1}{200}$  -  $\frac{1}{50}$  sub cutem, and light diet; sometimes by inhalations of amyl nitrite or subcutaneous injection of trinitrin or sodium nitrite, as vaso-dilators of the peripheral blood vessels.

For dyspepsia try a mixture of arsenic, sod. bicarb. and tr. gent. co. or infus. gent. co. after food; for vomiting, bismuth carb. or cerium oxalate, etc.; for diarrhœa, salol and large doses of bismuth. During fever the child must be kept in bed. If there are night sweats sponge with weak vinegar and water, give food and stimulants shortly before the sweating usually begins, and possibly atropin, agaricin gr.  $\frac{1}{24}$  -  $\frac{1}{6}$ , or other drugs such as are used in adults.

Attention to detail is of the utmost importance in chronic phthisis. In generalised tuberculosis and the tuberculous forms of pneumonia and broncho-pneumonia treatment is carried out on general principles, such as are adopted in capillary bronchitis and simple broncho-pneumonia.



## CHAPTER LXII.

### SYPHILIS: ACQUIRED AND CONGENITAL.

Syphilis is due to the *treponema pallidum*, or *spirochata pallida*, a spiral motile organism discovered by Schaudinn (1905). It is best seen in fresh preparations, from the edge of a sore or mucous plaque, by means of the ultra-microscope. Using Giemsa's method of staining, avoiding nuclear stains, it has been found in the cut sections of many organs in congenital syphilis, especially the liver. Levaditi (1906), with silver nitrate staining, found it most often and most numerous in the liver, lungs, adrenals and skin in descending order, and absent or scanty in the brain and kidneys. It has been found in the umbilical cord, spleen, lymph nodes, blood, and the bullæ of syphilitic pemphigus. It is mainly present in the organs most often affected histologically. In acute, rapidly fatal cases it is widely distributed; in chronic ones it is chiefly in the liver. Although it is spread by the blood it is rarely intra-vascular. It quickly makes its way through the walls of the vessels, becomes perivascular, and multiplies in the cellular tissue. The anatomical changes are periarteritis, endarteritis, infiltration of connective tissue with mononuclear cells and secondary sclerosis, and degeneration of parenchymatous cells. The transmission of the organism and the effects of its toxin produce dystrophies as well as immediate pathological changes.

**Acquired Syphilis.**—The disease may be acquired in early life by infection during labour, if the mother has secondary or primary syphilis of the natal passages; and after birth from one or other parent, a wet nurse or attendant, or by accidental contact. *Coincident* syphilis in mother and child is due to infection of the mother about the time of parturition, and secondary infection of the child. Infection is conveyed directly by kissing, sexual contact, infected instruments, or in the Jewish mode of circumcision; indirectly by spoons, teats, feeding utensils, thermometers, toilet apparatus, etc.

The primary sore is usually extra-genital, most frequently on the lips, less often the mouth, tonsil, eyelid, cheek, face, neck, etc. It may be a mere papule, with no characteristic induration. A vulvar chancre is most likely to be of criminal origin. The disease may be milder than the congenital form for it appears later, when the child is better able to withstand its influence. The rash is usually macular, scanty and evanescent, often absent. Condylomata are frequent and sometimes present in huge numbers



on the mouth, pharynx, nose, genitals, anus and scrotum. The glands are enlarged generally. It runs the same course as in later life but is more acute. It is much more contagious than the congenital type, and must therefore be diagnosed early. Probably many cases of late syphilis, ascribed to the congenital disease, are really due to the acquired form. The evidence of acquired syphilis in infancy is a chancre appearing at least 21 days after birth and secondary symptoms after about 45 days. Secondary symptoms in the first 2 months of life and lesions such as snuffles, pemphigus, epiphysitis and cranial bossing, are signs of congenital syphilis.

**Congenital Syphilis.**—There is no true heredity of syphilis. The disease is acquired during intra-uterine life, or the separation of the placenta, by transmission from the mother. Yet healthy children have been born though both parents have been infected. Probably many inherit the taint and never show symptoms, growing up healthy. The question constantly arises whether a woman can have a syphilitic child without being infected herself. In other words, can the infection be transmitted directly by the sperm cell to the ovum, the mother escaping infection or acquiring immunity from the foetus in utero? Or is the child invariably infected through the placental circulation, as suggested by the finding of syphilitic changes in the placenta?

*Conceptional Syphilis.*—Many women have borne syphilitic children and have remained under observation for years without developing specific sequels. They have either been rendered immune by impregnation or, much more probably, have been very mildly infected. The absence of signs of infection is not proof of non-infection. Lesions, supposed to be typically syphilitic, may be found in children whose parents give no evidence or history of the disease.

*Sperm Transmission* is most improbable. The *treponema pallidum* is 1-4 times the diameter of a red corpuscle in length, and it is difficult to believe it could become entangled with the impregnating sperm cell and thus infect the ovum. Beneficial results on the offspring are produced by anti-specific treatment of the father only, the mother being apparently healthy. Recurrence of the disease in the father may be followed by the birth of a syphilitic infant, healthy ones being born when he is properly treated. Possibly this means that the nutrition of the sperm cell is impaired by the disease, or that the organism is conveyed in the semen to the vagina and thence to the uterus or blood of the mother, finally reaching the foetus. The organism has been found in large numbers in a 4½ months foetus (Bar and de Kervilly, 1907). Finger (1903) collected 16 cases in which the mother of syphilitic children on a subsequent marriage to a healthy male bore healthy children. In such instances it is probable that the mother was only mildly infected and the effects had died out. Paternal transmission is improbable, except through infection of the mother.



*Maternal Transmission.*—The mother can transmit the disease when the father is unaffected. Inoculation experiments on apparently healthy women who have borne syphilitic children have proved negative. Even though a woman has several affected infants, she may remain in apparently good health, and she can nurse her syphilitic infant without contracting the disease though the child is potentially capable of infecting a wet nurse (*Colles's Law*). According to this law every mother of a syphilitic child is immune. This does not prove that she has been infected during pregnancy nor that she has acquired immunity from the foetus without being herself infected. There is no evidence that the mother can be infected by the foetus in utero. Absorption per vaginam of an immunising substance in the sperm is a remote possibility. Exceptions to Colles's law, chiefly in primiparæ, have been recorded but are of doubtful value. A man had syphilis 6 years before marriage. His first child was breast-fed, had syphilis and died in the third year. The second child, born 19 months after the first, had a papular eruption in the mouth and was breast-fed. The mother acquired a hard chancre on the nipple and a secondary rash (Novy, 1906). Such a case is strongly in favour of germinal paternal infection. As far as I know it is unique. Intra-uterine infection is most likely to occur in the early months of pregnancy, before the placenta is differentiated. Later on the placenta must be sufficiently diseased to permit the transmission of the organism. A child born within a dangerously short time of the primary disease in the parent may escape. As a rule the later the stage of the disease in the parent, the less severe is it in the infant. A common sequence is one of miscarriages at later and later stages of pregnancy, the birth of still-born infants, then of others with the disease, and finally healthy babies. The sequence may be irregular, perhaps on account of recrudescence of the disease in a parent or of treatment. One child may suffer very definitely and all the others escape. It is unusual for several to suffer in succession, probably because of treatment. In a few instances one of twins has been affected and the other healthy.

It is not infrequent to find a first child healthy and a later one syphilitic. Although this may depend on parental recrudescence of the disease, it must not be forgotten that it may be due to syphilis acquired by the father during the wife's first pregnancy or confinement, by the mother subsequently, or by the child through transmission from another male progenitor. The only symptoms may be malnutrition and arrest of growth. The absence of early signs is no proof that the child will be exempt from those of late congenital syphilis.

Disease in the mother does not necessarily imply either disease or immunity in the child. According to *Profeta's Law* the healthy child of a recently infected mother is immune; that is, it is syphilitic, but presents no evidence thereof, or there is a germinal transmission of immunity. On the other hand many such infants have become infected. Such immunity is



only temporary and does not persist beyond puberty. Re-infection increases the virulence of the disease and its effects on the offspring. The syphilitic mother of a non-syphilitic child can nurse her baby with little risk of transmitting the disease.

*Contagiousness of Congenital Syphilis.*—The discovery of the treponema is strongly in favour of the infectivity of the disease. Clinical evidence affords little support. Many of the older writers regarded the congenital form as highly contagious, much more so than the acquired disease. In 1898 Bulkley considered it a constant source of the acquired disease. Fournier and Sir J. Hutchinson, probably the two greatest authorities on the subject, hold that it is infective and may cause nipple chancre in a healthy wet nurse. Henoch has not found one wet nurse become infected. Günzburg, Physician to the Foundling Hospital, Moscow (1872), stated that 31 healthy wet nurses suckled 120 syphilitic children for a total period of 395 months and remained perfectly healthy. Each nurse was in the syphilitic ward for 6-10 months. Most recent writers regard it as little, if at all, contagious. Coutts has only seen one doubtful case. Still has not seen one, nor have I.

Colles's Law has been accepted as proof of infectivity. But the fact that the mother does not acquire the disease from her child is no proof that a wet nurse will do so. In almost all cases of supposed infection it is difficult to exclude the possibility of the infecting case being one of the acquired disease. The following instance (W. R. Grove, 1906) illustrates this. On second-hand evidence the mother was said to have had syphilis 2 years before the birth of her illegitimate child. She returned to service soon after. The child had universal pemphigus shortly after birth and recovered. It was treated for 4 months. At 8 months it had condylomata; and at 18 months condylomata and stomatitis. Then the grandmother and 2 children, aged 12 and 7 years, developed primary sores on the tonsils. Infection was supposed to have been carried by feeding utensils. But it is possible that the infant had acquired syphilis, or that the family were infected from another source. A universal pemphigus, ending in recovery, cannot be accepted as proof of congenital syphilis.

There is, however, proof of infectivity in the discovery of the treponema and in the transmission by vaccination. Cory had to make use of 4 children before he was able to infect himself with the disease. Hutchinson collected a number of cases in which persons acquired syphilis by vaccination with lymph from affected infants. It must be admitted, unless we assume that in all these instances the infant was suffering with acquired syphilis, that the disease can be transmitted, though its contagiousness is slight and has been much over-estimated. The organism is not always present in the local lesions, blood or lymph.

*Syphilis in the Nurse.*—In view of the possibility of a wet nurse claiming that she has acquired the disease from the infant, it is important to



consider the evidence *pro* and *con*. A chancre takes at least 21 days to develop and secondary symptoms appear in about 45 days. Hence, the stage of the disease may be too advanced for it to have been caught from the child. The position of the primary sore should be conformable, e.g. on the breast. There must be further evidence such as induration, a bubo, mucous plaques, rash, condylomata, etc. It must be quite clear that the child showed priority of infection. Either the child or the nurse may have acquired the disease from each other or independently. Examine the nurse's child and husband. If the child is unaffected, the nurse has not had recent syphilis. The nurse may have been giving the breast to other infants or may have acquired a mammary chancre from venereal contamination.

*Syphilis of the Third Generation.*—It is generally accepted that the children of those who have had congenital syphilis are not affected. Before regarding any case as affording reliable evidence of transmission to the third generation, many sources of error must be taken into account. Either parent, though congenitally syphilitic, may have acquired the disease in later life. Both primary and secondary syphilis may be mild and overlooked. The husband is not necessarily the father. Boeck (1904) reported 3 cases. The grandmother had secondary syphilis; the mother congenital syphilis in 1860 at 2 years of age, and many signs thereof in 1889 when she brought her child, aged 4 months, with typical congenital syphilis. The mother showed no evidence of the acquired disease, had had no miscarriages, and had had a healthy first-born child. The husband gave no evidence of syphilis. Possibly the father was syphilitic or the mother had been re-infected after the birth of her first child. In the second case the grandmother had acquired syphilis in 1873. The mother showed scars about the mouth in 1897 and had a congenitally syphilitic child. The father was not examined. In a third instance the grandmother showed tertiary syphilis, the mother had scars about the mouth and Hutchinson's teeth, and her first 2 children were syphilitic. Here too the possibility of re-infection of the mother or a syphilitic father was not excluded. In a case recorded by C. F. Marshall the evidence of congenital syphilis was weak and the father was not examined. The grandmother had scars of gummata about the knees. The mother, at age 16, had gummata in the popliteal space and extensive destruction of the palate. Her first child had saddle nose and frontal bosses, the second died of bronchitis, and the third was healthy. There were no miscarriages.

Fournier collected 116 cases of which 59 in his opinion fulfilled the conditions required. He states that the disease manifests itself by dystrophic stigmata like those of the second generation, and by virulent syphilis in 14 per cent. Tarnowsky (1904) agrees with Fournier as regards the transmission of dystrophic effects but not of virulent syphilis. Possibly a dystrophic influence is transmitted in the form of a tendency to prematurity, delicacy and malnutrition. As a rule congenital syphilis has



ceased to be virulent by the time procreative age is reached. Fournier investigated the results of the marriages of 46 patients with congenital syphilis. Out of 145 pregnancies there were 39 still-borns, 43 abortions and 63 living children. Nearly all showed stigmata of degeneration, especially defects of the eyes and teeth. According to him certain diseases may be syphilitic in origin but not in nature. To this condition he gives the name of "*parasyphilis*." Syphilitic affections are closely allied to and simulate non-specific ones. There is no strict line of demarcation. The so-called parasyphilitic affections are anæmia, marasmus, rashes, choroiditis, interstitial keratitis, leucoderma and notched teeth. They can occur independently of syphilis and may be unaffected by specific drugs. Parasyphilis, as a distinct affection, needs more evidence in its favour.

On the whole we must regard transmission of the disease to the third generation as not proven. There is not the least evidence of such transmission when the transmitter of the second generation did not have congenital syphilis.

**Pathology.**—A diffuse blood infection sets up a diffuse cell proliferation, i.e. an inflammation, in the perivascular connective tissue of the minute blood vessels. The lesions are multiple, similar in type, but modified by the structures in which they occur. Blood vessels may be obliterated by contraction of the perivascular connective tissue or by secondary endarteritis, spreading from without. The organs are much increased in weight but may show no macroscopic evidence of disease. The parenchyma is sometimes unrecognisable. There is a tendency to cirrhotic changes, and to aggregation of newly-formed cells in masses as syphilomata. Minute areas of anæmic necrosis, the size of a large pin's head, are found in the liver, kidneys, adrenals and epiphyses. The liver is always affected in the foetus and generally in all infants. The skin, lungs, pancreas, kidneys, adrenals, spleen and epiphyses are attacked. The inflammatory changes in the skin start as a proliferation in the blood vessels of the sweat glands. According to some authorities the intestinal glands of the foetus, formed in the third and fourth months of pregnancy, first exhibit this diffused syphilitic inflammation. The blood signs are deficiency of red cells and hæmoglobin, the presence of nucleated red cells, and a leucocytosis, especially of myeloblasts and eosinophiles. Secondary arrests of development are liable to occur in affected organs.

**Symptomatology.**—*Fœtal syphilis* is a visceral and bone affection. Hepatic enlargement and osteochondritis are the chief signs in the foetus and still-born child. The bone changes, especially at the epiphysial lines, are the most constant. There is a tendency to diffuse infiltrations in the viscera of the foetus and the cutaneous tissues of the infant, notably the palms and soles. The changes are most marked in the structures which, at the time of infection, are undergoing the most rapid development. The skin is of somewhat later development, as regards its glandular structures, than



other organs and is consequently affected in the later months of pregnancy, shortly before birth, or not until after birth. Although its glands are formed at the fifth month, their blood vessels do not develop until the seventh month of foetal life. The palms and soles are particularly liable because of their multiple sweat glands.

*Syphilis at Birth.*—Pregnancy may end in the expulsion of a macerated foetus, a dead foetus, a still-born babe, a premature and feeble infant which survives a few hours or days, a babe showing signs of the disease, or one which is apparently healthy. Death of the foetus is most common between the fourth and seventh months, and is due to placental disease or severe general infection. Though maceration of the foetus is not conclusive of syphilis, it is strongly suggestive for it is due to this cause in about two-thirds of the cases.

The presence of signs at birth is indicative of grave infection. The babe is feeble, dull, torpid and ill-nourished, with wrinkled skin, old-age aspect and subnormal temperature. It usually exhibits an eruption of papules, pustules or bullæ, snuffling, jaundice due to intercellular hepatitis, enlargement of the liver and spleen, epiphysitis and perhaps choroido-retinitis. Death ensues in 1-4 weeks.

*Later Signs.*—Frequently there is no indication at birth and the symptoms appear during the first two months of life, less often in the third month and rarely later. In about half, the signs appear in the second to fourth week. They are most marked in the skin and mucous membranes. Mild cases are often unrecognised.

*Wasting* is an important sign and, if present in the first three months without an apparently reasonable cause, is very suggestive. It may be of a very severe type, beginning shortly after birth, with no definite relationship to the severity of the syphilitic signs and not improving as these signs subside. There may be no demonstrable changes after death. Wasting, in proportion to the severity of the symptoms, is of less grave prognostic import. At birth the child may be a fine baby. If breast-fed and not gravely infected it may maintain its nutrition. More commonly it is not so fed, or the breast milk is insufficient in quality and quantity, and it wastes progressively until it reaches the stage of marasmus in which the facial aspect is that of wrinkled old age, with the skin hanging in folds all over the body and deficient in elasticity, in colour like that of *café au lait* through infiltration and pigmentation of the rete Malpighii, and in texture thin and dry. The child is whining, irritable, peevish, restless and sleeps badly and may be feverish from enteric disturbance, epiphysitis, etc.

*Rhagades.*—The lips are stiffened by infiltration and crack at the angles, forming radial fissures. Similar fissures are found at the edges of the nostrils and eyes and in the genito-anal region. The *tongue* has a shaven appearance the filiform papillæ being stripped off, the fungiform



left, and the circumvallate enlarged. Papules and mucous plaques are seen on the buccal mucosa.

*Snuffles* (p. 374), a subacute or chronic coryza, is present in  $\frac{2}{3}$ - $\frac{3}{4}$  and is one of the first signs. It usually begins before the rash in the first month, commonly in 2-3 weeks, and may be present at or shortly after birth. It gives rise to mouth-breathing, loud snuffling and difficulty in suckling. In severity it varies from slight stuffiness up to acute obstructive hypertrophic rhinitis, ulceration of the nasal mucosa and necrosis of bone.

In babies the discharge is primarily serous, later yellow and irritating, and may become profuse, muco-purulent and bloody. It may only be visible in the pharynx. The nose is blocked by swollen mucous membrane and thick crusts. The bridge becomes thickened and depressed, the tip swollen and tilted up, and its appearance describable as "pug nose" or "saddle nose." The pug nose is due to contraction of the tissues and cartilage, and the nostrils look upward. Sometimes the bridge never develops although there has been no definite external evidence of disease and bone destruction. Snuffles must be differentiated from other causes of nasal discharge (p. 371).

Its course is chronic, more than 2 weeks. The catarrh frequently extends to the pharynx and larynx, interfering with swallowing and respiration, and affecting the voice.

Gummatous deposit in the submucosa and periosteal thickening may lead to ulceration, caries and extensive necrosis, even in the early months of life. It is more frequent in later childhood and causes ozæna, destructive ulceration of the nasal structures, necrosis and falling in of the nasal bridge. The nose may be almost destroyed in a few weeks.

*Rashes.*—The skin eruption is more or less synchronous with the coryza, usually a few days to a week later. It may come out independently if the coryza is absent, slight or overlooked. It is present in two-thirds, uncommon after 6 months, and exceptionally delayed until the end of the first year. The common sites are the soles and palms, the glabella or triangular space between the eyebrows, the bathing drawers area, the legs, trunk and arms, and the muco-cutaneous junctions. Itching is rare. Polymorphism is a marked characteristic. Spots tend to coalesce into extensive areas and in situations like the napkin region are liable to superficial ulceration. Secondary pus inoculation may occur. Many varieties, diffuse or circumscribed, have been noted independently or in combination, and occasionally associated with non-specific rashes, such as erythema, intertrigo, seborrhœa, lichen urticatus, eczema and pustular dermatitis. The chief varieties are :—

1. Simple erythema of the buttocks and perineum, extending down the inner side of the thighs, on to the scrotum or vulva, and even on to the abdomen and down to the feet. This rash is not evidence of syphilis, unless it distinctly spreads beyond the area of friction (bathing drawers area),



affects the palms and soles, and is smooth and scaleless. The tint is variable.

2. A dry desquamative rash is characteristic. It particularly affects the palms and soles, causing the epidermis to peel off in great flakes and leave a smooth, dusky red, glazed or glossy surface of sclerosed and thickened skin.

3. The common rash is macular or papulo-macular in type, such as is seen in acquired syphilis, and sometimes pustular. The macular rash is the mildest and the pustular one the most serious. The rash is often polymorphous. Macules are more or less circular, disc-shaped patches of a roseolous, pale rose red, dusky red, raw ham or coppery colour. They appear on any part of the body but have a special predilection for the junction of the forehead and hairy scalp, the glabella, cheeks, chin, neck, buttocks, hands, feet and the flexor surfaces of the upper and lower extremities. There is evidence that a roseola never appears on the trunk in congenital, only in acquired syphilis. Papules are most frequent on the palms and soles. The macules become covered with fine scales. Finally smooth coppery discs are left and persist for some time. They may desquamate, or fade from the centre, and leave pigmented spots. The rash develops fully in 1-3 weeks and lasts 1-2 months.

A frequent combination is a diffuse erythema of the bathing drawers area, with roseolous macules, papules or coppery discs on the adjacent skin and distributed fairly generally over the rest of the body, least on the trunk. The rash is never as bright red as true intertrigo, and the skin feels stiff from the infiltration but is not swollen. The scrotum is often swollen, œdematous and peeling. Condylomata are fairly common. Ulceration is rare except in moist areas.

4. Pemphigus is a bullous rash with a special affinity for the palms and soles where it first appears. It is not seen in acquired syphilis. It is a sign of severity and may be present before, at or shortly after birth, and is rare after the fifth week. The bullæ are as big as peas or, by coalescence, reach the size of marbles. Their contents are sero-pus or bloodstained serum. The base is infiltrated, excoriated and copper coloured. Ulceration and even gangrene of the skin may supervene. On the face and head the rash becomes pustular. Papules appear on other parts. It must be distinguished from pemphigus neonatorum (p. 142), a rash which is never present at birth and has no infiltration of the bases of the bullæ.

5. Secondary ulceration of the mucosa, usually superficial, is apt to occur and spread to the skin or the thickened skin is liable to crack and fissure (rhagades). These ulcers are multiple, linear, bleed readily, and form fissures radiating outward from the angles of the mouth, nose, eyes and anus. They leave behind radiating scars.

6. Mucous patches and condylomata are papules modified by peculiarities of situation. Mucous patches are found on the buccal mucosa, tonsils



and soft palate. Condylomata develop from papules situated in moist contiguous places such as the anus and perineum, folds of the buttock, scrotum, vulva, behind the ears and sometimes in the axillæ. They are raised, flat-topped, moist, whitish discs,  $\frac{1}{4}$ - $\frac{1}{2}$  in. diameter. They are rarely seen before 6 months of age; may be found in the genito-anal region from the second to the sixth year, rarely after the fourth year; and are much more frequent in the acquired disease. Both mucous plaques and condylomata are apt to end in superficial ulceration, especially in the mouth and genitals.

7. Ringed eruptions, vesicles, pustules and serpiginous rash are uncommon varieties. Ulcers are occasionally deep and serpiginous. The infiltration of the buttocks may be dense and gummatous.

8. Cutaneous gummata, sometimes described as "blind boils" or a "furuncular syphilide" (G. Carpenter), are by no means rare during the first year. Apparently they are not a true gummata. The skin is dusky, coppery or bluish, and contains nodular infiltrations. These attain the size of peas, are raised above the surface, and may have a vesicular, pustular or scabbed apex. Occasionally they are larger or aggregated into groups. They often break down by central softening and necrosis, discharging their contents and leaving a small deep ulcer which soon heals. They are most common on the head, buttocks and thighs.

9. Subcutaneous gummata are rare in infancy but may occur at any later age. They are often single, at first movable, and form dome-shaped, projecting, dusky red or purplish, rather soft nodules of variable size. Later they adhere to the skin which is infiltrated, hyperæmic, and breaks down, forming a deep, clean cut or serpiginous ulcer. The usual site is the head or the neighbourhood of a joint. A white scar is left on healing. Muscular gummata are very rare.

*The Hair and Nails.*—A profuse growth of hair, straight, dark in colour and fine in texture, has been regarded as specific and named the "syphilitic wig." The evidence in its favour is inconclusive. Usually the hair becomes very thin and scanty in a few months, especially on the vertex and eyebrows. Permanently bald patches may develop. Absence of occipital hair is due to many causes and not of diagnostic value. Absence of eyebrows is more suggestive. Eruptions on the scalp may stimulate sebaceous activity and the formation of seborrhœic crusts. The subjacent skin does not ulcerate and patches of the rash may be seen at the edges of the crusts.

The nails may show a *paronychia sicca vel ulcerosa*, or a true suppurative inflammation of the matrix, generally in conjunction with a desquamative rash. The adjacent skin at the bases is discoloured, thick, glossy, scaly and crusted. The nails become discoloured, opaque or dusky; shrivelled, furrowed, arched or claw-like, or narrowed at the base and fan-shaped at the free edge; dry and brittle; and may be exfoliated, perhaps repeatedly.



A transverse furrow or ridge divides the new, more or less healthy nail, from the thin soft diseased portions. The finger nails are more affected than the toes.

*The Bones and Joints.*—These are secondary rather than tertiary symptoms, multiple, symmetrical, early in onset, and transitory. Epiphysitis, a cause of pseudo-paralysis, is present in 10-20 per cent. It is described together with the later manifestations of joint disease, in a previous chapter (pp. 817-8). *Periostitis* may occur independently of epiphysitis and form nodes, rarely under 6 months of age. The humerus and tibia are chiefly affected in infants, and the tibia in older children. It is more frequent about puberty, at which age osteitis may occur. The diaphysis becomes spindle-shaped from thickening of the periosteum and the joint may be involved. Subsequently it leaves a thickened, rounded tibial crest and an antero-posterior sabre-like curvature, chiefly of the middle third. Fournier has consequently named the tibia the *os revelateur*.

*Dactylitis* may affect the fingers and, to a less extent, the toes. It is a gummatous periostitis and the interior of the bone is unaffected or sclerosed. It occurs in the first few years of life, even as early as the third month. It begins in and chiefly affects the proximal phalanges, especially the index finger, rarely attacks the metacarpals and metatarsals, and never involves the joints. In a girl, 3 months old, the first metatarsals were alone diseased. It may be limited to one finger, or be multiple and often symmetrical. The affected parts are painless, swollen, red and fusiform. The inflammation is subacute, tends to recovery and does not break down. It must be distinguished from *tuberculous dactylitis*, closely resembling it in appearance, due to a caseous focus in the bone and secondary periostitis, and tending to suppuration. In this the metacarpals and metatarsals may be affected.

*Gummatous cranial syphilis* is uncommon in early childhood. Hochsinger saw 4 cases in 600 syphilitic nurslings. It ends in absorption, the formation of a cranial boss, ulceration, or necrosis and exfoliation, perhaps leaving a large gap in the frontal or parietal region. *Parrot's nodes* is a name given to the cranial bosses or hyperostoses of the skull, due to excessive formation of bone in the frontal and parietal regions surrounding the anterior fontanelle, giving the skull a "hot-cross bun" aspect, the sulci being the sutures. The forehead is square and upright, the frontal eminences prominent, and the bones very hard. These bosses develop after birth. They are frequently absent in syphilis, present in rickets with no evidence of syphilis, and still more often absent in rickets. Possibly they are rachitic in origin (p. 184), or the result of syphilis as an additional factor. They must be regarded as specific if they are present in the first few months of life in a child with a small anterior fontanelle and hard bone. The nodes have a bluish colour as seen through the thin scalp. Frontal bosses and a prominent square forehead are rachitic. *Craniotabes* has been described



as a rachitic sign (p. 184). The type in which there are numerous, small areas of softening, crackling on pressure, chiefly in the parietal bones, in the first 4 months of life is probably syphilitic in origin.

*Necrosis* leads to depression of the bridge of the nose, exfoliation of the vomer and turbinals, perforation or destruction of the septum nasi and hard palate, and destruction of the cranial bones. Perforation of the nasal septum has occurred at 6 months (Goldreich, 1908). These effects are rare before the onset of the first dentition and are commonly delayed until about puberty. Most of them are secondary to gummatous infiltration.

*The Circulatory System and Blood.*—Developmental errors (p. 466) may be present. Duguy ascribed many cases of mitral stenosis, of which several were associated with other defects, to this cause. Myocardial and endocardial changes, gummatous aortitis, and endarteritis are late affections. Arteriosclerosis may be due to the specific toxin. Endarteritis is rare in the viscera; it may be found in the brain and in chronic meningitis, and cause softening.

Interstitial myocarditis may occur in infancy and cause sudden death while in apparent health. It is indicated by paroxysmal dyspnoea, cough and intense cyanosis. Foci of anæmic necrosis in the heart are liable to be mistaken for gummata.

Anæmia is proportionate to the severity. The blood picture is that of secondary anæmia or of a more infantile stage of development. Severe anæmia, independent of splenic hyperplasia, may appear in the second year, without other evidence of syphilis or after the symptoms have subsided. Hæmorrhages arise from ulceration of the mucous membranes. Umbilical bleeding depends on vascular disease impairing contractility. Some cases of hæmorrhagic disease of the newborn (p. 127), and possibly hæmoglobinuria, are syphilitic in origin.

*The Glands and Spleen.*—The glands in the axillæ, groins and occipital region are often palpable and a little enlarged, hard, separable and movable; usually in conjunction with anæmia and enlargement of the liver and spleen.

The spleen is palpable in about half the cases but clinically should not be accepted as definitely enlarged unless it extends  $\frac{1}{2}$  in. below the costal margin. Marfan (1903) agrees with Birch-Hirschfeld that the spleen of non-syphilitic stillborn babies is 0.33 per cent., and that of the syphilitic is 0.76 per cent. of the body weight. Hochsinger gives the relative proportions as  $\frac{1}{325}$  and  $\frac{1}{198}$  of the body weight. Marfan states that 75 per cent. of enlarged infantile spleens are due to syphilis and almost all the rest to rickets. Out of 376 infants under 2 years old 40 had large spleens:—undoubted congenital syphilis 18, probable 13 (with rickets in 12), rickets without syphilis 6, tuberculosis 1, and no definite cause 2. The hyperplasia is an interstitial splenitis with fibrous overgrowth, chiefly affecting the



Malpighian bodies and vessel walls. Perisplenitis is not uncommon and may cause adhesions.

*The Respiratory System.*—Catarrhal laryngitis is a common early symptom, causing hoarseness and aphonia, and is liable to be mistaken for croup. The chronic form is generally a late symptom and adult in type. It causes chronic cough and hoarseness, perhaps aphonia, and occasionally attacks of dyspnœa which may be so severe as to necessitate tracheotomy. The various parts are swollen or ulcerated. The epiglottis may be deeply fissured, the ary-epiglottic folds ulcerated and the vocal cords thickened. Some cases have condylomata or papillomata on the epiglottis and vocal cords. Older children may show perichondritis, papillomatous excrescences, ulceration of the larynx, destruction of the uvula and epiglottis; scarring, ulceration, gummata or perforation of the palate; gummatous affections of the larynx and trachea, and even tracheal fistula. Acute laryngitis, œdema of the glottis and laryngeal stenosis may occur. A case of Frankl's died at 3 months from acute stenosis due to necrosis of the cricoid cartilage and left arytenoid. Mattosso (1905) reported stenosis at 4 months of age. From birth the boy had stridor, increasing difficulty in breathing and nursing, retraction of the chest, suffocative attacks and wasting. Recovery followed injections of biniodide of mercury. Stenosis of the trachea or bronchus is a rare sequel of gummatous infiltration. The symptoms (p. 393) are uninfluenced by posture.

In the foetus the lungs may show gummata or white hepatisation, the "*white pneumonia*" of Virchow, a fibrous induration with grey areas of consolidation. Microscopical examination reveals infiltration with lymphoid cells; cylindrical or cubical masses, the remains of foetal lung tissue; and fatty degeneration of the alveolar septa. Its general appearance is much like sarcoma. Possibly the supposed gummata are remnants of foetal lung tissue. Gummata have occasionally been found in the first year of life and later. They may soften and produce bronchiectasis. Some of the chronic fibroid changes, interstitial pneumonia, in the lungs of infants are possibly of syphilitic origin. Both fibroid changes and gummata may be present.

*The Alimentary System.*—Apart from mucous patches and rare faucial affections, there is nothing characteristic of the disease in infancy. Subsequently the tongue may show rough, corrugated patches, suggestive of leucoplakia and perhaps due to recurrent condylomata; and in later childhood may be enlarged, thickened and contain indurated nodules, or a single unilateral gumma. Deep ulceration of the palate, pharynx and naso-pharynx is rarely seen in the first year. It usually occurs about puberty, is followed by caries and necrosis, and is remarkably rapid and destructive in its progress.

The milk *teeth* show no special changes. They may appear unduly early and are prone to decay. Hutchinson described certain peculiarities of the



permanent teeth, notably a semilunar erosion of the upper central incisors, and peg-like lateral incisors. The central incisors are the most liable to show defects. They are dwarfed, rounded and peg-like, short and strong; and sometimes present a central crescentic notch and chisel shaped section, a concave free edge, bevelled anterior surface and rounded corners. The deformity is present before they are cut. It is due to impaired nutrition of the dental germ from various causes. The bicuspidis are often peg-like. The first molars are "reduced in size and dome-shaped, through the dwarfing of the central tubercle of each cusp" (H. Moon, 1884); or show 4 yellowish cusps or protuberances which are excavated and not covered by enamel (Suker, 1905). These are the first teeth of the second dentition to calcify and the most liable to be damaged by mercurial stomatitis in infancy. The other teeth escape unless mercury is given in later infancy, and then the first bicuspidis may be affected (Hutchinson) and horizontal furrows form across the incisors and near the apex of the canines. Fournier attaches importance to a *horizontal white streak* on the surface of the incisors. Enamel defects are not always due to congenital syphilis or mercury, and the absence of such defects cannot be relied on as evidence of non-mercurial treatment in infancy (p. 223). Sound first molars are in favour of no such treatment. The malformation of the upper central incisors is the one dental defect to which importance can be attached as a sign of congenital syphilis.

The *alimentary tract* may show perivascular hyperplasia in the mucosa, causing diffuse thickening and injury to glandular structures. The *pancreas* may present an indurative enlargement, a sclerosis with fatty degeneration of the cells and obliteration of the lobules. The *liver* is the most frequently affected visceral organ. According to Hochsinger the relative weight of the liver to that of the body is 1 : 21·5 normally, and 1 : 14 in congenital syphilis. It is large, soft at first, and later uneven and indurated (p. 365). The indurative type is sometimes associated with jaundice and, less often, with ascites, splenic enlargement and engorgement of the abdominal veins. It responds better than the spleen to anti-syphilitic remedies.

*The Genito-Urinary System.*—Nephritis is acute interstitial, interstitial and parenchymatous, or chronic interstitial. Areas of anæmic necrosis may be present. An acute interstitial nephritis has been described by Coupland (1876), Stroebe (1891), and by Sutherland and Thomson Walker (1903). The interstitial connective tissue is infiltrated with round cells. Stoerk of Vienna (1901) maintains that syphilis retards the renal development, so that at birth the outer layer of the cortex contains imperfect tubules and glomeruli. Speiss (1877) collected 34 cases of the disease and found renal changes in 10. Possibly there is a diffuse perivascular connective tissue infiltration, causing imperfect development of the cortex, Malpighian bodies and tubules. Gummata are rare. Chronic interstitial nephritis (p. 581) has been ascribed to syphilis and cases in favour of this.



view have been reported by Massalongo, Guthrie and Carpenter. Superficial affections of the genitals (pp. 871-3) and orchitis (p. 596) have been described.

*The Nervous System.*—The developmental defects include hydrocephalus due to arrest of cerebral development with degeneration of the optic thalami, to meningitis or to ventricular ependymitis. It is rarely as great as in non-specific cases, may be present at birth or comes on in the first 3 months of life, and is incurable. Other defects are spina bifida; and congenital club-foot, due to atrophy of the cells of the anterior cornua and Clarke's column. *Pachymeningitis*, simple or hæmorrhagic, and cortical sclerosis may occur in the first year of life and be associated with gummata. It is less rare at 6-15 years of age, and gives rise to headache, mental deterioration, fits, wasting and death from asthenia. *Meningitis* is rare and ill-defined, diffuse and basal; localised, chronic and gummatous, perhaps hæmorrhagic. Ventricular ependymitis is the most common sign in infancy, giving rise to rapidly increasing hydrocephalus, squint, tremor and convulsions. Cerebral arteritis is common at the base of the brain and in the small vessels of the brain and cord. Both arteritis and meningitis give rise to encephalitis and myelitis, with softening or secondary sclerosis and atrophy, and imbecility. Hemiplegia may depend on local encephalitis or gummatous meningitis, but is rarely syphilitic. Gummata are rare in the brain and cord; less so in meningitis and inflammation of the cranial nerves. Barlow reported gummatous neuritis of several nerves at 4 months of age. The third, fifth and seventh are most liable to be affected.

The onset and progress of cerebral syphilis are subacute, and the symptoms multiple and complex. One variety usually predominates at the onset and throughout, e.g. Motor signs such as tremor, athetosis, palsies, rigidity and affections of speech and the cranial nerves; Visual symptoms, especially Argyll-Robertson pupils, squint, optic neuritis and atrophy, and choroido-retinitis; or Psychical phenomena, viz. changes in disposition, diminished intellectual power, backwardness, irritability, depression or excitement, and causeless fits of grief or anger. Insomnia and sensory symptoms are rare. Headache, occasionally hemicrania, is common, early, and not severe. Temporary disorders of speech or vision, vertigo and sudden syncope, may depend on circulatory disturbance. Restlessness, irritability, sleeplessness and fits may be due to slightly increased cerebral tension or hydrocephalus in the eruptive period. The fontanelle is then tense or bulging.

Either the congenital or acquired disease may give rise to cerebral syphilis as above described, epilepsy, Jacksonian epilepsy, cerebral palsy, cerebral sclerosis, multiple sclerosis, tabes, general paralysis of the insane, backwardness, imbecility and idiocy, spinal palsies, myelitis and rarely multiple neuritis. Grinker (1904) reported a good illustration of the transmission of nervous instability. The mother had syphilis 4 years before marriage and developed the first symptoms of tabes 9 years after the birth



of her third child. The first child, a boy, had typical juvenile tabes at 15 years ; the second, a boy, had general paralysis during adolescence ; and the third, a girl, showed signs of cerebral syphilis soon after birth. Intervals of 2 years occurred between each birth.

*The Eyes.*—Iritis is occasionally seen in the first 18 months and is bilateral in about 50 per cent. It is said to occur even in utero but the evidence thereof is unreliable. It is not uncommon in late congenital syphilis as a complication of keratitis. It is simple, mild and slowly progressive, perhaps with exudation ; or gummatous, with small yellowish nodules at or near the margin of the iris. *Interstitial keratitis* occurs at 6-15 years of age, sometimes earlier or later. It is common, most frequent in the eldest child and in females, and insidious and painless in onset. The child may be in excellent health. It begins in the ciliary body of one eye and affects the other in a few weeks. Occasionally one recovers before the other is attacked. The cornea has the appearance of ground glass and the episcleral vessels are injected. Later, vascularisation of the cornea gives it a salmon, cherry red or dark red colour. Pain, photophobia and blepharospasm are present ; ulceration and suppuration are rare. About two-thirds to three-quarters of these cases are due to congenital syphilis and the remainder to acquired syphilis, malnutrition or tuberculosis. Under efficient treatment recovery is generally complete, but may take months. The opacity may become white, dense and persist as a leukoma. *Choroido-retinitis* is present in 15 per cent. in the first year (Still). It is usually discovered accidentally unless specially sought for, and is not proof of syphilis, for it occurs independently. In one variety there are white or pale yellow patches of atrophy, often surrounded or spotted with pigment. In another, the condition is like that of retinitis pigmentosa and often associated with nyctalopia. *Optic neuritis* is rare, atrophy more common.

*The Ears.*—Otitis media, secondary to the pharyngitis, may become chronic and cause deafness. Acute attacks, sometimes painless and apyrexial, end in perforation of the drum and otorrhœa. It is a cause of deaf-mutism. It is doubtful whether specific inflammation occurs in infancy. *Bilateral deafness* of rapid development is much more common in girls than boys, aged 8-25 years. It is due to disease of the auditory nucleus, nerve or labyrinth. The common type is a chronic osteitis or periostitis, causing more or less complete occlusion of the cavities of the internal auditory meatus and labyrinth, just as in the tertiary form of acquired syphilis. This sets up an acute or subacute inflammation of the nerve or its terminals. It begins insidiously, without pain or vertigo, in one ear and later on in the other. Complete permanent deafness may result in a few weeks. If the labyrinth is affected the onset is somewhat acute and there is vertigo for the equilibrating apparatus is involved, perhaps at first by increased tension from effusion. Incurable tinnitus may be present. It is less common than, and frequently precedes, though it may follow, keratitis.



**Diagnosis.**—Fœtal syphilis can be recognised by osteochondritis, demonstrable by X-rays after the fifth month of intra-uterine life. Infantile syphilis may be very easy or extremely difficult of recognition. Diagnosis is based on the aspect of the child, the symptoms and physical signs, and to a certain extent on the response to mercurial treatment. A family history of miscarriages and still-births is suggestive but not conclusive. Evidence of the disease in older children is of more value. Much tact must be exercised in eliciting the family history. To ask the father whether he has had the disease may, if he has not had it or is unaware that he may have been infected, lead to unwarranted suspicion of the mother. The absence of evidence of infection in the maternal history or the previous children does not exclude the possibility of recent infection. On the whole it is of little use troubling much about a history. A negative one is valueless, a positive one is not conclusive proof that the particular illness is of syphilitic origin. Other diseases occur in syphilitic children and there is much too great a tendency to ascribe such diseases, when present, to the specific taint.

Valuable points in diagnosis are wasting without obvious cause, the association of snuffles and rash, anæmia, and splenic and hepatic hyperplasia. Epiphysitis is almost conclusive.

*Late congenital syphilis* is equivalent to the tertiary stage of the acquired disease. The ulceration of the skin and mucous membranes, and the affections of the bones and joints, must be differentiated from those of tuberculous origin. The late signs develop after the third year, usually at 6-15, and most commonly at 12 years of age. Stigmata in the form of radial scars at the angles of the mouth, etc., are the most reliable sign of past disease. The general signs are a greyish earthy pallor, sallow skin, anæmia, delicacy, malnutrition, physical and mental backwardness, dwarfism, infantilism, and a delay of 1-4 or more years at puberty. These children walk and talk late. The natiform or hot-cross bun type of head, deformed nose, Hutchinson's teeth and curved tibia may be present. The chief active signs are keratitis, progressive deafness, hyperplastic osteitis and periostitis, joint affections, destructive ulceration of the nose, naso-pharynx and larynx, gummata and gummatous enlargement of the liver or spleen.

A sunken nasal bridge is strong evidence, and the destruction of the soft palate, perforation of the palate or vomer, and ulceration of the epiglottis and larynx are practically certain proof of the disease. Gummata are conclusive. They chiefly affect the tibia, bones of the upper limbs, sternum and cranium. They have been found in the third year but are rare before puberty. It is often difficult to be certain whether the late signs are due to congenital disease or syphilis acquired in early life.

**Course and Prognosis.**—Congenital syphilis after birth must be looked upon as the secondary stage of the disease acquired in intra-uterine life. Usually it is progressive, except in so far as the skin affections tend to



become less marked. Mucous patches may recur in the mouth up to the sixth year; and condylomata, especially in the genito-anal region, not infrequently recrudesce, even repeatedly. An imperfectly treated rash may return in the second year. Specific intracranial affections may arise independently of other evidence of infection. Many of the symptoms described might be spoken of as relapses, but are rather evidence of the persistence of infection, although there are periods of latency in which the special symptoms are absent.

The outlook depends on the severity at birth. A puny, marasmic infant with a pustular or bullous rash is almost certain to die. Much wasting in the first 3 weeks is a bad sign. The earlier the symptoms appear, the worse is the prognosis. Late rash is a sign of mild infection. Marked enlargement of the liver and spleen is unfavourable. Visceral lesions at birth are of bad prognosis; skin and bone affections are curable. The degree of nutrition, mode of feeding, nursing and hygiene influence the result. A well nourished, breast-fed infant, who develops mild symptoms in the second month of life, will recover. An ill-nourished, bottle-fed baby with severe symptoms in the second or third week will probably die. Apart from the immediate effects of the disease, the child is more liable to rickets, bronchitis and broncho-pneumonia, intestinal disorders, and septic infection. Its vital resistance is low. It readily contracts infantile disorders and dies from them, and is liable to convulsions. Often it presents developmental defects incompatible with life or decreasing the chance of existence. The prognosis is worse in hospital than in private cases for there is less likelihood that the mother has been treated during pregnancy. The later infants have a better chance for they are less severely affected. Early and prolonged treatment will completely cure many children. Recrudescences may occur between infancy and puberty. The prognosis in the acquired disease is generally favourable.

Choroidal atrophy does not tend to get worse. Late affections are curable if prolonged treatment is adopted sufficiently early. Recovery from keratitis, choroiditis and, less often, deafness, is good and usually permanent. Nervous complications and sequels are more serious; the outlook being best in gummatous, worse in arterial, and very bad in degenerative lesions.

**Treatment.**—Prevention is better than cure. No syphilitic man should marry until at least 2 years have elapsed since the appearance of active symptoms. Hochsinger insists on efficient treatment, 4 years interval from the primary infection, and entire freedom from signs for 6 months. The interval should be even longer in women. The mother must be treated during pregnancy if she has recently acquired syphilis, if she has had it some considerable time previously but no recent treatment, if there has been any recrudescence, or if the father has had the disease within 2 years previous to the mother's conception.



If the child is healthy at birth the risk of transmission of the disease to an attendant is trivial. Syphilitic parents must be warned that there is a risk and proper precautions adopted. Maternal nursing is essential if practicable; otherwise recourse must be had to artificial feeding.

If there is strong reason to suppose infection give a course of treatment for 4-6 weeks. Otherwise wait for the first symptoms. Blue ointment is the most reliable preparation. Oleate of mercury 1, ung. rosæ 4 parts, or mercury vasogen, may be used. Wash the child with warm water and soap, dry, and rub in about grs. 15, a lump of ointment the size of a pea, for 5 minutes nightly into the skin of the abdomen, chest or limbs. Or spread a larger quantity on a binder, from the ensiform to the pubes, and leave it on for 2 days. If the skin gets sore, apply a simple ointment, and rub the blue ointment into the axillæ or groins. In addition give grey powder gr.  $\frac{1}{2}$ -1, with aromatic chalk powder, t.d.s. Add to this Dover's powder gr.  $\frac{1}{8}$ - $\frac{1}{4}$ , if there is any diarrhœa; or bismuth. carb., cretæ prep.  $\overline{aa}$  grs. 2-4. Other suitable mercurials are liq. hyd. perchlor. m. 3-10; Van Swieten's fluid (hyd. perchlor. 1, alcohol 100, aq. destill. 900) m. 3-10; hyd. protiod. (yellow iodide); liq. hyd. perchlor., with pot. iod. gr. 5 in each drachm; tannate of mercury; and calomel gr.  $\frac{1}{10}$ . Hochsinger prefers the yellow iodide 1 part in pulv. acaciæ 50 parts, as more efficacious than grey powder or Van Swieten's fluid, and states that it rarely causes transient diarrhœa. Calomel produces rapid effects but is likely to set up diarrhœa. For severe pustular rashes a daily bath of hyd. perchlor. grs. 5-10 in 5 gallons may be used. On the whole mercurial inunction and grey powder are the safest measures and generally sufficiently efficacious.

In all cases watch for gangrenous stomatitis and cancrum oris. Keep the mouth clean. Salivation is absent for the glands are not developed. The duration of treatment should depend on the severity of the case. Continue it energetically until symptoms have disappeared, and gradually omit it in the course of the next 3-4 months. A case of medium severity requires full treatment for 2 months; grey powder only for another 2 months t.d.s.; then twice and finally once a day. It must be repeated if there is the least relapse. In the late manifestations it is essential to get the child as quickly as possible under the influence of mercury. Mercurial plasters are good, but liable to cause troublesome skin irritation. Daily injections of biniodide of mercury (gr.  $\frac{1}{12}$ ) for 2-3 weeks at a time act rapidly. Intra-muscular injections are unsuitable for children, painful and unnecessary, but have proved successful.

Grey oil, 40 per cent. strength, is injected in doses of 1 c.cm. for the newborn deeply into the buttock under strict antisepsis every 8-15 days. A long, firm platinum needle is used. The induration, if any, is said to disappear quickly. I have no experience of this treatment. Iodides are necessary in late stages, and occasionally useful in infants, especially if



there are gummata. From gr.  $\frac{1}{2}$ - $1\frac{1}{2}$  per year of life can be given daily in water and milk.

For sores on the buttocks and mucous patches on the vulva apply calomel 1, zinc. oxid. 1, amyli 2-4 parts; iodoform, or lotio nigra. Use pure calomel for condylomata and mucous warts; ung. hydrarg. ammon., 33 per cent. strength, for ulcers. The latter ointment is beneficial for nasal sores, being inserted into the nostrils after removal of crusts. Or cotton tampons covered with red precipitate ointment are placed alternately in each nostril for an hour at a time, after cleaning with a cotton swab. Or the yellow oxide of mercury ointment, 1 in 8, can be used. Adrenalin drops relieve nasal obstruction. Phagedænic ulcers of the nose or palate are cauterised with nitric acid or the acid nitrate of mercury. For gummatous ulcers apply ung. hyd. amm., ung. hyd. oxid. rub., or lotio nigra, and cover with sublimate gauze. For interstitial keratitis prescribe a shade, fomentations and an ointment of cocaine gr. 5, atropine gr. 5, vaseline oz. 1; and mercurial inunctions or intra-muscular injections, together with pot. iod. internally. Arsenic and extract of thyroid gland are beneficial in some cases. Pilocarpine injections gr.  $\frac{1}{20}$ - $\frac{1}{8}$  may do good in early stages of deafness.

The maintenance of the general health by syr. fer. iod., ol. mor. and other tonics is a factor sometimes more conducive to cure than treatment by specific remedies.



## CHAPTER LXIII

### DIPHTHERIA.

Diphtheria is a specific infectious disease with a local lesion in which the specific bacillus is found. The infection is almost always in the upper respiratory passages and the local mischief only mechanically dangerous to life. The systemic effects on nervous and muscular tissues are due to soluble toxins. The bacillus was found by Klebs in 1883 and isolated by Löffler in 1884. Subsequently Roux and Yersin separated the toxin.

The disease is regarded as primarily local, because of the mild cases in which it produces few or no constitutional effects. It spreads through the blood and lymph channels to internal organs. It is regarded as primarily constitutional, because of its incubation period of 1-7 days; the early occurrence of albuminuria or nephritis, within 24 hours in malignant cases; and the cases of severe and fatal nephritis and profound systemic infection although the local disease may be trivial.

The affection is faucial, pharyngeal, laryngeal, nasal, and exceptionally in other situations, such as the conjunctiva, vulva, skin or wounds. Membranous sore-throat is not invariably diphtheria for it may be due to a pneumococcus, streptococcus, staphylococcus or yeast. Often there is a mixed infection. The presence of the specific organism in the false membrane is conclusive, but its absence is inconclusive. Negative results are of comparatively little value. A swab may have been badly taken, few bacilli may be actually present, their vitality may have been weakened by recent antiseptic applications, and they may fail to grow on the culture media. If a case is clinically diphtheria, it should be treated as such and notified, although the bacteriological report is negative. The organism usually disappears from the throat of convalescents within 6 weeks of the onset.

**Bacteriology.**—Three kinds of organisms are generally recognised, though as many as 20 morphological varieties have been described. The true bacillus is Gram-positive, produces acid in glucose broth, and stains readily with Neisser's stain. It is a slender rod, uniformly cylindrical or slightly curved, dotted or beaded, sometimes club-shaped, and non-motile. The characters vary with the culture media. It is easily destroyed by heat and chemicals, but resists cold and drying. Hofmann's bacillus, or the pseudo-diphtheria bacillus, is often present in normal throats. Some assert that it is an attenuated form of the true bacillus and that its virulence can be increased by successive passage through small birds, rendering it pathogenic.



to guinea-pigs and rabbits. More probably it is a totally different organism. It is innocuous to animals producing no pathological lesions, forms no acid in glucose broth, and can be distinguished by its morphological, cultural and biological features. A third group consists of much less virulent diphtheria bacilli found in healthy throats or in convalescence, especially during epidemics, their lack of virulence being due to some anti-action of the tissues or secretions. The bacilli vary in virulence in different cases. They are widely distributed, and possibly some additional factor is required for their development. The toxin causes local necrosis, destruction of red cells and diminution of hæmoglobin, subnormal temperature, primary nerve degeneration and paralysis, and degeneration of cardiac nerve centres and muscle fibres. The membrane is due to the toxin, for it can be produced by injection of germ-free toxin. It is somewhat doubtful whether diphtheria without membrane formation exists.

**Mode of Infection.**—The disease is spread by direct contact, sputum, infected hands and articles, and by animals. Little is known of the life of the bacillus outside the body. The disease is more common in rural districts and isolated farmhouses than in towns. It has become more prevalent in towns in recent years. Probably the germs can be propagated independently of the sick, when they are deposited in suitable places. In New York City the drains were foul and insanitary conditions prevalent, yet there was no diphtheria before 1850. After that the organism was introduced and now the affection is widespread. Improved sanitation appears to have little effect. It is very contagious, though the contagion is limited to the immediate neighbourhood of the patient. It may be limited to one side of a class room, if it is separated from the other by a fairly wide passage; to a single house, street or parish; and spreads very slowly. Mild ambulatory and unrecognised cases are the main source of its spread, especially where the population is dense and aggregated in schools, churches and places of amusement. The school habits of licking slates, pencils, and sucking the same sweets are very dangerous. Children may be “carriers” of infection though themselves uninfected. Infective organisms can be carried long distances in trains, cabs and other conveyances and by garments or articles of merchandise. Big outbreaks have been traced to infected milk. Diphtheria in the cow is generally, if not always, due to human infection.

*Avian diphtheria* affects pigeons, fowls, turkeys, pheasants, partridges, quails, parrots and sparrows. A big epidemic occurred in the Thames Valley district in 1907. The prevalence of an offensive membranous sore throat in pigeons has been followed by an outbreak of malignant diphtheria in the inhabitants of the district, probably through eating the pigeons. Possibly the infection may be carried by eggs for false membrane has been found in the oviducts of fowls, ducks and pheasants. Cats, sheep and pigs have suffered from a disease of the throat in several localities in which diphtheria was prevalent. Cattle, horses, dogs and rabbits are also liable.



Some of these epidemics are not due to the Klebs-Löffler bacillus, but are caused by the bacillus of necrosis, protozoa or by organisms producing septicæmia. Calf diphtheria and that form common in lambs are due to the bacillus of necrosis. It must, however, be accepted that cats, rabbits and birds may get diphtheria and convey the infection. Or the cat may be merely a "carrier."

**Pathology.**—The toxin is allied to snake venom in character. Ehrlich maintains that it is composed of 2 substances :—(1) Toxin, to which antitoxin is an antidote, causing acute symptoms of poisoning ; (2) Toxone, causing degeneration of peripheral nerves. Sidney Martin has shown that the bacillus secretes a toxin ; and can digest proteins, forming albumoses and an acid body, both poisonous. Others maintain that only one poisonous substance is produced. There are two stages of the disease, *toxic* and *paralytic*, with no line of demarcation between them. The paralytic may be regarded as due to the action of the toxin rather than to an independent poison. The toxin produces fatty changes in the diaphragm, heart muscle, kidneys, adrenals and liver, and to a less extent in other organs. Later on the vagus, phrenics and other nerves are affected. Antitoxin neutralises the toxin and will prevent this fatty protoplasmic change, if it is given early. The pathological change is in active progress by the fourth day. Fatty change in the heart muscle may be found on the third day as the cause of acute cardiac failure. It antedates any nerve affection. The heart shows no macroscopical change and no "tabby-cat" striation (Dudgeon), but minute granules of fat are visible on staining with Sudan III. or Scharlach R. In later stages of the disease the heart shows interstitial changes. The muscles undergo hyperplasia, swelling, loss of striation and fatty change. Fatty changes may be found in the diaphragm before the heart, although there are no symptoms. In the kidneys it chiefly affects the convoluted tubules, and there is no interstitial or vascular change, even though there is albuminuria. The liver is extremely prone to fatty change and may even float in water. Dudgeon (1908) has also shown that extensive fatty changes occur in the adrenals, causing deficient production of adrenalin with early acute congestion and hæmorrhage in guinea pigs, as well as fatty changes in the medulla. In cases fatal from acute toxæmia degenerative changes have been found in the motor nucleus of the vagus and in the anterior cornua of the spinal cord, as well as cardiac degeneration. In later stages there is usually considerable change in the peripheral nerves.

The local condition is a coagulation necrosis of the epithelium due to the toxin, vascular engorgement of the mucosa, exudation of cells, and coagulation of the exudate. Hence the membrane is composed of coagulated fibrin, leucocytes, epithelial cells, debris and bacilli in groups or nests. If quite superficial, it can be peeled off. More often it involves deeper structures, and secondary necrosis causes ulceration or gangrenous changes.



**Etiology.**—A familial susceptibility has been noted. The predisposing factors are lowered vitality from any cause, abrasion or impaired vitality of the epithelium, and catarrh of the mucous membrane. It is especially apt to follow measles, scarlet fever and whooping cough. Meteorological conditions are unimportant, except as a cause of catarrh. The disease is rather more common in girls, perhaps because they are fond of pets. It is rare in infants under 6 months, most common at 2-10 years, and causes the greatest number of deaths in the third and fourth years. Nasal diphtheria has been recorded at 2 weeks of age (Kühn), and faucial diphtheria on the eighth day of life (Auden, 1902).

**Symptoms of Faucial Diphtheria.**—The onset is insidious with general malaise. Epistaxis, as a prodromal symptom, indicates gravity. Vomiting is a rare initial symptom. Occasionally the onset is acute, with throat symptoms, T. 101-102° F., and the usual signs of fever. Severe attacks may be ushered in by rigors, vomiting or convulsions, and a temperature up to 104° F. or more, or with marked prostration. Very extensive membranous inflammation of the fauces may exist in a child, without the least complaint or discomfort. Sometimes there is pain in the neck and on swallowing. Pallor, offensive breath, listlessness, depression and languor are often marked early signs. There is complete disinclination for food or play, with fretfulness and irritability, and the voice may be hoarse or nasal.

The appearance of the throat at the onset and throughout is sometimes that of follicular tonsillitis. There may be little or no exudation, a little cheesy follicular deposit, or small necrotic areas on the tonsils. The membrane may start so low down on the tonsils as to escape detection. Usually there is seen on one or both tonsils, or on the pharynx, patches of membrane which rapidly spread and coalesce. The membrane is tough, adherent, and leaves a bleeding surface if detached. It varies in colour from a greyish white to dirty yellow or even black. It is smooth, flattened, and depressed below the surrounding mucous membrane, which overlaps it as the rim of a watch-glass overlaps the glass. It tends to spread toward the air passages and on to the uvula and soft palate. A primary patch on the uvula is almost certainly diphtheria. The membrane indicates the intensity of the local process but is no measure of the toxæmia. It peels off or is liquefied by the action of antitoxin in 24-48 hours. In severe cases, there is great faucial œdema, much membrane and horrible fœtor. The tongue is covered with a thick, creamy fur. The tonsils and uvula are greatly swollen, red and œdematous; and there is general extension, with much swelling of glands and subcutaneous tissues (angina Ludovici). The submaxillary lymph glands are usually a little enlarged, not to the great extent so often seen in follicular and other forms of tonsillitis. There is frequently pain in the ear on the affected side.

In pure faucial diphtheria the mucous membrane is rather pale, not inflamed, and there is no muco-purulent secretion. The child looks ill,



with a pale leaden aspect and small frequent pulse. Albuminuria is slight or absent, T. 100-101° F. for a short time, and the glands are a little or not at all enlarged. The chief danger is that of extension to the larynx and bronchi. If antitoxin is injected there is marked improvement on the following day, local recovery in 2-3 days, and convalescence in a week.

In diphtheria associated with streptococcal or staphylococcal infection the mucous membrane is red and sometimes bleeds, the inflammation limited or generalised, and the secretion muco-purulent. The temperature ranges from 101-104° F. The breath has a saprophytic odour. The tonsils are always swollen, glands almost invariably enlarged, and the nose generally attacked. The membrane is not characteristic but may be very thick and extensive, if due to staphylococci. It is apt to give rise to broncho-pneumonia, otitis, and septicæmia.

**Pharyngeal Diphtheria** is localised, follicular, spreading, or malignant. The milder types give rise to dysphagia, occasionally angina Ludovici, and constitutional symptoms, and usually get well under antitoxin treatment in a week. The larynx generally escapes. Sometimes anæmia is extreme and ulceration extensive. The malignant type is rare under 4 years of age. It is due to excessive virulence, mixed infection, or increased susceptibility. It produces extensive and deep lesions with severe toxæmia. It may gradually supervene on the localised or spreading variety, or be ushered in acutely with fever, vomiting and epigastric tenderness. The skin is dusky, features bloated or drawn, and the neck and pharynx swell enormously. The mucous membrane exhibits intense redness, much membrane and mucus, with foetid odour and black tongue. The mental condition is one of restlessness, excitement, insomnia and slight delirium; or prostration and apathy. The temperature is low, perhaps high at the onset, and albuminuria is present.

**Laryngeal Diphtheria** may be due to gradual extension but more often comes on suddenly about the fourth day of the illness, or is primary in the larynx. It may be limited to the ventricles of Morgagni. The early symptoms are those of simple laryngitis, with less fever and more general depression. A gradual or sudden onset is followed by all the signs of increasing laryngeal obstruction, viz. hoarse or muffled voice; tickling cough, becoming dry, harsh, brassy, hollow or barking; inspiratory stridor; and attacks of asphyxia. In these the child thrusts its fingers into its mouth, tears at the throat, and becomes livid, convulsed, drowsy and comatose. Or they are milder in type, lasting for a few seconds, with anxious look, staring eyes, sweating forehead, cyanosis and crowing inspiration. Casts of the trachea and bronchi may be coughed up. The symptoms may become acute in 24 hours, or severe obstruction may not develop for some days. It terminates in asphyxia with cyanotic pallor, pinched nose, drawn features, sweating forehead, cold extremities, rapid superficial breathing, thready pulse and ominous calm.



**Nasal Diphtheria**, sometimes called *membranous* or *fibrinous rhinitis*, is primary or secondary. It often follows scarlatina. Nine-tenths of the cases are under 10 and few under 3 years of age. In some cases there is no membrane, although pure cultures of the bacillus are obtained. In others the symptoms are mild and yet there is much membrane. At first the discharge is acrid, clear, watery and irritating, perhaps sanious. Later, it becomes muco-purulent or purulent, and may contain shreds of membrane. Sometimes it is peculiarly offensive. It is unilateral in about 20 per cent. It causes redness and excoriation of the *alæ nasi*, congestion and nasal obstruction, and often epistaxis. In a day or two there may be found a white film or a tough gelatinous yellowish membrane in the anterior nares. This may be limited to the anterior end of the septum or cover the whole of the mucous membrane; or limited to the naso-pharynx and found only on the choanæ and orifices of the Eustachian tubes. Finally, only blood and pus are exuded.

A mild type, limited to the nose, gives rise to slight constitutional symptoms, such as, pallor, languor, anorexia and moderate fever. The glands are rarely enlarged. It runs a benign chronic course, rarely causes epistaxis or sepsis, is only mildly infectious but may spread the disease in schools. The organism does not seem to produce enough toxin to impair the general health to any great extent. A more severe type is generally secondary to faucial infection. A gangrenous form begins in the nose with œdema, swelling, shiny overlying skin, marked pallor, fœtid secretion and epistaxis. Renal and nervous complications are uncommon in nasal diphtheria, and the disease rarely spreads to the pharynx and larynx. It is not followed by paralysis, although it lasts from 1-3 months. In the newborn it causes nasal obstruction, asphyxia while suckling, anæmia, prostration and adenitis.

**Cutaneous Diphtheria** follows some excoriation or injury. It may be wide-spread and very severe. A definite membrane with surrounding zone of inflammation is present, or a gangrenous slough with subjacent ulceration, not at all like the diphtheritic process and only diagnosed by bacteriological examination. Vulvar diphtheria is similar in type.

**Conjunctival Diphtheria** is most frequent under 4 years of age, and is either primary or due to extension up the lachrymal ducts. It begins on the palpebral conjunctiva. Typical membrane is formed or the appearance is more like gonorrhœal ophthalmia, with patchy deposit on the surface. It may spread to the bulbar conjunctiva, involve the cornea, and cause destruction of the eye.

**Aural Diphtheria** is due to extension to nasal passages and then up the Eustachian tubes, occasionally to the tympanum and mastoid; or the orifices of the tubes are blocked by swelling. The otitis is very painful, causes violent headache, sometimes delirium or coma, and rapid tympanic necrosis.



**Diphtheritic Stomatitis** is rarely primary. Thin, greyish white patches, like aphthous stomatitis, are found on the mucosa and may become confluent and thick. It may begin on the lips and spread to the tongue and larynx. More frequently it is secondary to faucial infection and may spread even to the skin. The oral cavity is lined with membrane, salivation increased and foetor marked. Membranous stomatitis is sometimes due to other organisms.

**Hæmorrhagic Diphtheria**, confined to children, is a malignant variety with severe bleeding from the nose, mouth and pharynx; into the stomach, intestines and urinary tract; and into the skin. A purpuric rash during convalescence, or epistaxis or faucial bleeding only, is not sufficient for such diagnosis. It is most common in cases in which early treatment has been neglected. They react less readily to antitoxin. It causes vomiting, foetid diarrhoea and profound anæmia; and death from coma or convulsions, in a few hours or days, in over 80 per cent.

**Hypertoxic Diphtheria** causes heart failure, cyanosis, unconsciousness and death from toxæmia, although the local signs are slight.

**Latent Diphtheria** is a name given to cases with no local lesion or sign of ill health, viz. "contacts"; cases with local lesions and no general symptoms; and those in which there is anæmia, indefinite malaise, and some rhinitis, otorrhoea, faucial catarrh, cutaneous sore, etc., which is bacteriologically diphtheritic. It is an unsuitable name, except for the first variety.

**Toxæmia** is indicated first by an irregular and intermittent pulse, with or without signs of cardiac dilatation, and progressive cardiac failure; diminution in the amount of albuminous urine, occasionally anuria; vomiting, without nausea and independent of food. It comes on early, usually in the first week while membrane and adenitis are still present. The child becomes greyish, pale and cold, listless, and refuses food. Temperature falls and the pulse fails, although there may be no signs of obstruction to breathing and local manifestations may be subsiding. Drowsiness often comes on, though consciousness persists to the end. Restlessness is common, delirium an occasional symptom, and sometimes there are convulsions or coma. Breathing may be sighing or laboured, and pulmonary œdema is not infrequent. The symptoms are due to the action of the poison on the heart, motor nerve cells in the spinal cord, and the vasomotor system. It is probable that the rapid fall in blood pressure, collapse and death are due to the deficient secretion of adrenalin, from the action of the poison on the adrenal glands. The pulse rate is a valuable measure of the toxæmia, especially if it is out of proportion to the fever. The prognosis becomes steadily worse as the rate exceeds 100 per minute.

**The Heart.**—Endocarditis and pericarditis are very rare, while affections of the myocardium and its innervation are common and serious. The toxin



acts on the cardiac muscle and nerves. The earliest sign is irregularity. Later there develop dilatation, cardiac murmurs, gallop-rhythm, weak action, short and weak first sound, reduplication of the first sound, approximation of the first and second sounds, vomiting, and occasionally epistaxis, subcutaneous hæmorrhages, collapse and sudden death. Heart failure may occur in the acute stage of bad cases, even without warning. The pulse must be watched very carefully, for it varies in frequency and regularity at different periods of the same day. Three fairly defined types of myocardial mischief are seen. The first is usually fatal in 2 or 3 weeks and is characterised by gallop-rhythm, vomiting, epigastric pain and tenderness. In the second variety, not often fatal, rapid regular or irregular action of the heart may last for months. It is increased by exertion and gradually becomes normal. In the rarest type the pulse, at the end of the second or beginning of the third week, becomes very infrequent and, when it falls to 40 per minute, is associated with signs of prostration. The heart sounds are weak and there is a moderate degree of dilatation.

Murmurs, sometimes loud and musical, are more frequent at the apex than base and are due to relative insufficiency of the mitral valve. They are of little value in prognosis and occasionally persist after recovery. Dilatation may be quite sudden. The onset of serious heart complications is nearly always in the first 3 weeks. Vomiting, restlessness, subnormal temperature and an unaffected mind are characteristic features. Heart failure and persistent vomiting rarely appear before the seventh day and are due to extensive fatty degeneration. In later stages a certain amount of dilatation is due to anæmia.

Thrombosis may take place in an auricular appendage, renal vein or cerebral vessel. Cardiac thrombosis is an occasional source of death. It causes slow heart failure, pallor, cyanosis, cold extremities, restlessness and precordial pain, probably due to dilatation. The pulse is small, thready, quick and irregular. Death takes place without loss of consciousness. The thrombus may contain the bacillus and various cocci, and may break down, giving rise to infarction. Sudden death may occur in convalescence or even mild diphtheria, after slight exertion or a good meal, either from syncope or vasomotor palsy. Cardiac paralysis is most frequent from 2-9 years of age. Its duration is variable; 1-14 days if due to vagus paralysis, but much more prolonged if there is actual myocardial degeneration.

**The Kidneys.**—The renal changes are a measure of the toxæmia which falls on the renal epithelium. Albumin is present in 30-50 per cent. of all cases, generally on the fourth to the seventeenth day. Its frequency varies in different epidemics. The quantity increases with the severity of the attack. It is more frequent in fatal than non-fatal cases, and in paralytic than in non-paralytic ones. It is present in nearly all tracheotomy cases,



gangrenous attacks, serious sepsis, and cases sufficiently severe to cause paralysis of the pharynx and otitis media. It is not caused or affected by the injection of antitoxin. In toxæmia the amount of urine is diminished in quantity, and that of albumin varies directly as the intensity of the toxæmia. In mild cases it may only be present for 2 or 3 days. Hyaline and granular casts are uncommon.

It is sometimes due to actual nephritis and may then end in uræmia. Nephritis is rare and hardly ever becomes chronic. Anuria is due to toxæmia or to degenerative renal changes. Suppression is generally fatal. Urobilinuria and indicanuria are almost constant, phosphaturia often well marked, and acetonuria fairly common. Severe cases, especially if there is acute nephritis, may give the diazo-reaction.

**Complications** are due to toxæmia, the spread of inflammation downward and its effects on the lungs, local mischief, and the action of the toxin on the nervous system. Epistaxis is a bad sign when it occurs early, on the fourth or fifth day, and is a warning of probable toxæmia and hæmorrhage into the skin. It is an initial symptom in malignant angina, and of grave import if it precedes coryza. It indicates severity of infection and liability to cardiac failure from toxæmia. Sometimes it is due to separation of the membrane or sloughing.

Skin rashes may occur apart from antitoxin treatment, usually after the third day. They are urticarial, diffuse erythematous or measlesiform, and last for a few hours to 2 days. Skin hæmorrhages are most common under 7 and exceptional after 12 years of age. They are due to toxæmia; in cases not treated early by antitoxin, and in severe faucial or nasal diphtheria. They come out on the fifth to the tenth day, and are purpuric in character, like large bruises or purple spots, especially on areas subject to pressure. They may be found on the trunk, extremities, face, neck and ears; and are almost always of fatal significance, if there are more than two or three. They are associated with the usual signs of toxæmia, persistent vomiting, cardiac failure, epistaxis and occasionally hæmatemesis.

The respiratory complications are glottic spasm, bronchitis, bronchopneumonia, collapse, gangrene and interlobular emphysema. Diphtheritic membrane may extend down the pharynx and œsophagus and affect the stomach, or the latter organ may be involved without any evidence of direct spread (p. 257). Renal and cardiac affections must be regarded rather as symptoms than complications, except perhaps nephritis which is rare. The liver is often enlarged in fatal cases; and shows fatty degeneration in the acute disease or congestion from cardiac failure.

Various nervous symptoms have been noted, such as ataxy due to neuritis or inco-ordination, tetany, hemiplegia and other forms of palsy. Optic neuritis is rare. Progressive debility without actual paralysis may prove fatal. Otitis media is not uncommon. Adenitis may result in abscess formation.



*Diphtheritic paralysis* is a sequel rather than a complication, though it may occur very early in the disease. If it begins during the toxæmic stage it is due to the toxin causing degenerative changes in the central nervous system. Changes have been induced in the motor cells by injection of diphtheria toxin into rabbits (Rainy, 1900). Subsequently the symptoms are ascribed to toxone. The toxin has been neutralised and the nerve cells have recovered, but the nerve fibres show parenchymatous inflammation, and the heart shows interstitial changes. Probably these conditions are more or less combined. Thus, the cardiac symptoms are due to impaired innervation of a fatty heart, in consequence of acute degenerative changes in cells of the lower nerve centres producing an irritative and a paralytic effect on the vagus. Later on, they are due to peripheral nerve degeneration and interstitial cardiac changes. Motor, sensory and sympathetic nerves are affected with a parenchymatous inflammation.

The paralysis is more common in children than in adults, and in males than females. Insufficient food, bad nursing and lack of care during convalescence are predisposing factors. It affects 10-20 per cent. of all cases, and 30 per cent. of severe ones, but there is no constant relation between the severity of the attack and paralysis. Nevertheless it is a toxic manifestation and varies directly as the dose of the toxin, the extent of the membrane and severity of the disease, both in frequency and fatality. It is most frequent after faucial attacks. In malignant cases it may come on almost immediately. Albuminuria is much more common in paralytic cases. The proportion of cases is high, if loss of knee jerks alone is taken as proof of paralysis. Antitoxin treatment has not diminished its frequency; and indeed it is probably more frequent, if less severe, because of the recovery of many more of the severe cases.

It begins insidiously 1-3 weeks after the local symptoms have subsided, rarely as late as the seventh week. The early signs are weakness, feeble and irregular pulse, tendency to cough and splutter when fed, regurgitation of food, nasal voice, impaired vision, tingling and numbness in the lower limbs and loss of knee jerk. At first there is merely a little hesitation in the performance of motor functions. Palatal palsy is the most common symptom, and the earliest in about two-thirds of the cases. It may be limited to the soft palate, half or all of which hangs down, is motionless, insensitive, and does not respond to electrical stimuli. This gives rise to the nasal voice, regurgitation of fluid through the nose, difficulty in swallowing and unintelligible speech. If unilateral, the fluid only regurgitates through one side of the nose and the uvula is drawn to one side.

Paralysis of accommodation is due to cycloplegia and occurs in most cases, though it cannot always be recognised because of the difficulty in testing young children. Jaeger type and needle threading are the best tests. It is bilateral, rarely comes on before the third or after the seventh week, and lasts from 1-6 weeks. Other ocular affections are amblyopia,



amaurosis, mydriasis, inequality of the pupils and convergent squint. Almost all the ocular muscles may be affected in turn. Exceptionally the pupils react to accommodation but not to light.

Difficulty in swallowing, due to pharyngeal paralysis, begins almost always in the first to the third week. Most cases in which the onset is delayed to the third week recover, but two-thirds of those of earlier onset die. Food can only be swallowed after repeated attempts which fatigue the patient, and nasal feeding is necessary. It partly accounts for regurgitation of fluid through the nose, entrance of liquids into the air passages and cough. It lasts for a few days to 6-8 weeks. In laryngeal paralysis the adductors are affected and cough is toneless. It only occurs after laryngeal diphtheria, and gives rise to aphonia, irritating cough and dysphagia.

The early disappearance of the knee jerk is a bad sign. It is often enfeebled without being actually lost, and occasionally it may be a little exaggerated at first. It usually disappears in 6-8 weeks, or at any time within 6 months, and returns gradually in from 1-12 months. The Tendo Achilles jerk is affected less frequently than the knee jerk. Usually both are completely abolished in paralysis, and their absence may be the only indication of the loss of motor power in the lower limbs. They may remain absent for long after recovery and on one side longer than on the other.

Paralysis of the extremities is seldom complete. It is ushered in by gradually increasing weakness, numbness and formication. The gait becomes uncertain and the patient has incomplete perception of what he walks on. There is difficulty in the dark and in going up and down stairs. The legs are more often and more severely affected than arms. Sometimes the palsy precedes the loss of knee jerk. It usually begins in the fifth to seventh week, and may begin in the second or be delayed until the sixteenth.

Inco-ordination may occur without paralysis, and amount to staggering suggestive of cerebellar disease. Children stumble and fall about, bruising themselves, for the knees suddenly give way under them. Ataxic symptoms, combined with loss of reflexes and ocular troubles, give rise to the form of pseudo-tabes. The hands are clumsy, awkward and drop things, or may exhibit tremors like those of paralysis agitans. Inco-ordination generally comes on when the knee jerks disappear and lasts for a few weeks. It is nearly always associated with more or less paresis and may occur first. Sensory disturbance may be absent.

Various other palsies occasionally occur. Head-drop is due to weakness of the muscles of the neck, and inability to sit up to weakness of the back muscles. Acute inflammation of the nuclei may cause permanent palsy of one or more cranial nerves. Sometimes the tongue, lips, cheeks and larynx are affected as in labio-glosso-laryngeal palsy. In rare instances the paralysis is mono-, hemi-, or paraplegic. Hemiplegia is generally due to a vascular lesion. Paralysis of the diaphragm and the intercostals is serious and often fatal.



*Bulbar Crises.*—Bulbar palsy is due to the action of the toxin on the bulbar centres and is a cause of cardio-pulmonary paralysis, dyspnœa and rapid death. It comes on within 6 weeks of the onset of paralysis. Guthrie has called the dyspnœic attacks “bulbar crises.” According to his description the premonitory signs are restlessness and apathy, a weak hoarse nasal voice, irregular sighing respiration, loose weak cough, accumulation of mucus in the air passages and rapid pulse. The crisis is a sudden and acute exacerbation of the above symptoms due to excitement, physical exertion or no apparent cause. It produces sudden and complete paralysis of deglutition, aphonia, alarming dyspnœa and extreme restlessness. The alæ nasi are distended, mouth open, pupils dilated, colour bluish and skin sweating. The respiration is gasping, sighing and irregular, and there is general accumulation of mucus. The pulse rate is 150 or more; T. 102-103° F. Sometimes there is violent vomiting which may relieve the symptoms; occasionally the diaphragm is paralysed. These attacks last for a few minutes to hours, are recurrent, and may end in death from exhaustion, syncope, asphyxia or cardiac thrombosis. The first attack is rarely fatal.

*Sensory Affections.*—Anæsthesia of the soft palate is common, occasionally it is preceded by hyperæsthesia. The lips, nose, cheeks and limbs may be affected. Frequently it does not extend above the knees and elbows, but it may be general and associated with analgesia. Pains in the calves are common. Tingling, pins and needles, and numbness may be felt in the hands and feet. The bladder, rectum and special senses are affected in the worst cases. Incontinence is due to the general state rather than to sphincter paralysis. It may occur for a few days in a transient form during convalescence from lack of controlling power. The muscles give the reaction of degeneration and there is augmentation of galvanic, and diminution of faradic contractility. Slight contractures may occur in the most severe cases.

*Diagnosis.*—Diphtheria is overlooked on account of forgetting the golden rule to examine the throat of every sick child. Early diagnosis is of extreme importance and very difficult. The bacteriological report may be misleading. In some of the worst cases the bacillus may not be found. Other exudates may simulate that of diphtheria. The faucial affection has to be distinguished from certain mouth affections (p. 219) and many varieties of sore-throat (pp. 231-233); and the laryngeal obstruction from that due to other causes. Secretion, as opposed to membrane, may be found on the uvula and soft palate in septic and ulcerative sore-throats. Follicular tonsillitis associated with laryngitis is almost invariably diphtheria. The scarlatinal sore-throat is liable to be erroneously diagnosed as diphtheria, if exposure to cold has caused temporary disappearance of the rash. As a rule diphtheria comes on insidiously without vomiting; while scarlet fever is ushered in violently with severe vomiting and high fever, and there is



more of an appearance of sloughing and ulceration of the tonsils, or vivid redness at the onset. If there is neither laryngitis nor adenitis, the pseudomembranous sore-throat of scarlet fever may be regarded as not diphtheritic unless the bacteriological report is to the contrary. As a general principle the presence of anæmia, depression and lassitude greater than the severity of the signs would account for, in any throat case, with moderate fever and slight adenitis, are strongly in favour of diphtheria. More especially is this the case if there is unilateral rhinitis, coherent and tenacious membrane limited to one tonsil, or extending on to the uvula and soft palate, and albumin in the urine. In mild and doubtful cases the clinical symptoms may be limited to slight nasal catarrh, faucial trouble and otorrhœa, with or without constitutional symptoms, such as anæmia, increased pulse rate and general malaise. Occasionally there is no sign of ill-health or local lesion, but the typical bacilli are found on bacteriological examination. Morphological characteristics are insufficient for certain diagnosis. Cultural and inoculation tests take 4-7 days.

*Primary laryngeal diphtheria* in young infants is very difficult to distinguish from acute laryngitis, for nothing special may be noted until there are signs of laryngeal obstruction. The laryngitis at the onset of measles is apt to be mistaken for diphtheria, and in the course of the disease may be due to secondary diphtheritic infection. The constitutional symptoms are more severe than in simple laryngitis, and the diagnosis may be confirmed by the presence of a faucial secretion or the coughing up of membrane. Though membranous laryngitis is not always diphtheritic, it is advisable that such cases should be treated with antitoxin at the onset.

*Diphtheritic paralysis* is diagnosed by the history of recent sore-throat, laryngitis or nasal catarrh; and the presence of anæsthesia and immobility of the soft palate, nasal voice, regurgitation of food through the nose, defective accommodation, loss of knee jerks, etc. It may closely simulate acute anterior poliomyelitis. In one case the two affections were apparently combined. The child had a history of recent laryngitis, the diphtheritic bacillus was recovered from the fauces, and there was some slight inequality of the pupils, a little palatal and pharyngeal palsy, and marked paralysis of the lower intercostals. Gradually the knee jerks disappeared and paralysis developed in the muscles of the neck, back and limbs. In the course of his illness he had dilatation of the heart, tachycardia, submaxillary adenitis and otitis media. He recovered with partial paralysis of both legs and was a good deal wasted.

*Multiple neuritis* due to typhoid fever, measles, scarlet fever, small pox, syphilis, beri beri, etc., and poisons such as lead, arsenic, alcohol, etc., is almost unknown in infants and rare in children. Atrophy, rigidity and deep-seated muscular tenderness are said to be characteristic of the alcoholic type and absent in diphtheria. In one case of the latter variety they were



all present. Paralytic chorea, cerebellar tumour and Landry's paralysis are possible sources of wrong diagnosis.

**Prognosis.**—Under antitoxin treatment patients get well in a week. Otherwise the disease spreads easily, then remains stationary for some days, and subsides in 2-3 weeks. Epidemics differ greatly in severity, and the mortality varies in different epidemics and with the mode of treatment. The reduction in mortality depends upon early diagnosis, the early administration of antitoxin, and a sufficient dosage. The older the patient and the earlier antitoxin is given, the better is the prognosis. The mortality is very small in children over 2 years of age treated by antitoxin on the first day of the disease. Under 2 there is great liability to septic complications and under 5 years to respiratory ones. In the first 5 years of life the mortality is 10-15 per cent.; in the next 5 years 5-6 per cent.; and then it falls to 2-4 per cent. Laryngeal diphtheria is the most dangerous type in infants because of the small larynx, the yielding character of its walls, the spread of the infection and membrane downwards, or capillary bronchitis and broncho-pneumonia. Tracheotomy is an added danger, for it only relieves the obstruction and the membrane may continue spreading. Large tonsils, adenoids and nasal infection increase the risk. The constitutional symptoms vary greatly in severity and are most serious in the secondary nasal and the malignant faucial types. Mixed infections are more severe than pure diphtheria, and staphylococcal more dangerous than streptococcal ones. The dangers and degree of toxæmia, mechanical obstruction and carbonic acid poisoning must be estimated.

The gravest symptoms are early vomiting, abdominal pain, much albumin, anuria, restlessness, apathy, signs of profound toxæmia and its effects on the heart and circulation, weak first sound, gallop-rhythm, frequent and irregular pulse, vagus paralysis, and paralysis of the intercostals, diaphragm and pharyngeal muscles. A slow irregular pulse is a very bad sign in the acute toxic stage. Death is generally due to cardiac or respiratory paralysis, toxæmia, broncho-pneumonia or asphyxia. Broncho-pneumonia and the cardiac complications each accounts for about 25-50 per cent. of the fatal cases. Toxæmia must not be confused with cardiac paralysis. It occasionally produces respiratory paralysis and breathing ceases for some time before the heart stops. Tracheotomy in a case of this kind was useless. If due to toxæmia death takes place during the first 2 weeks, occasionally during the third week, and never before the third day. The diazo-reaction and hæmorrhages are unfavourable signs.

Vomiting is a bad omen. The earlier it occurs the worse is the prognosis. It may be present from the onset, start at the middle or the end of the second week, or begin after some weeks. Occasional vomiting, about 7-10 days after injection of antitoxin, may be due to certain properties in the serum; to food getting into the larynx in consequence of palsy; or to partially detached membrane irritating the fauces. Persistent vomiting is



grave and comes on at any period. In early cases it is a measure of the toxæmia and in the first 2 weeks is nearly always a fatal indication. Frequently it is associated with cardiac symptoms. It is induced by food, usually accompanied by nausea, may consist entirely of bile, and may be conjoined with increased peristalsis and loss of sphincter control, making it impossible to give food per rectum. Occasionally it depends on uræmia, diphtheria of the stomach, or direct irritation from inflammation and fatty changes; or on vagus irritation, an incipient neuritis, in which case it should be associated with bradycardia.

Sudden death during convalescence is rare. It may occur as late as the third month, and during apparently normal progress, even without warning signs. It is generally caused by excitement, vomiting, getting up too soon, or strain. Usually there is irregularity of the pulse, and no patient is absolutely safe while the pulse remains irregular. Arrhythmia may last for a few days to 6 months, and a latent weakness of the heart muscle may persist for years and develop under strain. Fainting attacks with vomiting may occur for 3 or 4 years.

Other bad prognostic features are failure of the appearance of a rash after antitoxin treatment; progressive hepatic enlargement preceding and accompanying cardiac failure, partly due to congestion and partly to fatty degeneration; and a punctate rash on the knees (Marfan).

*The prognosis of diphtheritic paralysis* is good, unless there is early cardiac palsy. It is good in the localised and generalised varieties, and bad in the cardio-pulmonary type. Bulbar crisis is usually fatal. Involvement of the muscles of the pharynx, the intercostal muscles and diaphragm increases the liability to food getting into the larynx, asphyxia, inspiration-pneumonia and broncho-pneumonia, and the difficulty of getting rid of bronchial secretions. Unfavourable signs are paralysis of the cervical muscles and marked ataxia. No case is hopeless. There is no permanent palsy.

*Relapses* occur in 1-2 per cent. and are difficult to explain. A relapse comes on after the second week, with fresh membrane and milder symptoms and without complications. It must be diagnosed from late tonsillitis and scarlet fever; and from the throat condition or pseudo-relapse, due to antitoxin, which comes on within 2 weeks with rash on the tonsils and skin, adenitis, arthritic pain and fever.

**Treatment.**—The mortality of diphtheria has decreased from 40 to 10 per cent. since the introduction of antitoxin treatment. The disease is not fatal, if antitoxin is given on the first day.

*Antitoxin* is a true antidote, combining with the toxin as long as it is free and rendering it harmless. It must, therefore, be given early, for it has little effect if the toxin has become chemically fixed in the tissue cells. It exerts no bactericidal or regenerative action on the cells, and does not influence septic complications. If given late, it only neutralises the



uncombined toxin. In malignant cases the poison gets too great a start. The importance of administering it early is well illustrated by the reduction in mortality of post-scarlatinal diphtheria in the Asylums Board Hospitals from 50 per cent. to *nil*, and in laryngeal cases from 70 to 30 per cent., and the reduced frequency of tracheotomy. We have to be careful in estimating its value since epidemics vary in virulence. Many mild cases are diagnosed on bacteriological grounds, and sometimes only the very severe ones are treated by antitoxin. It is advisable to give antitoxin to children without waiting for a bacteriological confirmation of the diagnosis. A single culture does not give infallible results. If the case is slight, limited to the throat which is clearing up quickly as the result of local applications, and with few or no constitutional symptoms, it is not always necessary; but it requires much experience and considerable courage to decide against its administration, for mild cases may become severe and individual susceptibility varies. It *must* be used if there are any laryngeal symptoms. It has no injurious effect on the kidneys and does not prevent diphtheritic paralysis. The objections to its use are its cost, the opposition of relatives, and the production of serum disease.

Simon states that a temporary reduction of leucocytes in the blood after antitoxin injection is followed in 3-4 hours by an increase. If this increase does not occur and the number exceed that present before injection, it indicates failure of the serum to act and is a bad sign.

*Mode of Administration.*—Use serum not more than 3 months old of strength of 500 units to 1 c.c. Administration by the mouth or rectum is useless. Inject it slowly into the subcutaneous tissues of the abdominal wall or side of the chest under strict antiseptic precautions. It acts more quickly intra-muscularly and intra-venously but, if used in this way, it must not be carbolised. The dose should be large and in proportion to the type and severity of the case; for faucial diphtheria 3000-6000 units, for nasal and laryngeal 6000-9000. Give 2000 units only in suspicious cases, and larger doses still in virulent cases or if treatment has been delayed to the third day. The dose must be repeated in 24 hours if there is no improvement, and even before if the symptoms are getting worse, especially in laryngeal attacks. Half-doses may be given every 12 hours until the membrane is separated. Doses up to 30,000 units at a time have been given, but do not appear of more value than those recommended. More should be given within 10 days if there is any relapse, if toxic symptoms persist, or if diphtheritic bronchitis develops. Intra-venous injection of 20,000-30,000 units has been given by Cairns (1902) in malignant cases with toxæmia, considerable involvement of lung and a moribund state. After the fifth day of the disease antitoxin appears of little value. Caiger reported that the mortality of 3000 cases at the Brook Hospital, during 1896-1899 inclusive, rose from *nil* in those inoculated on the first day to 3·6 per cent. on the second day, 6·7 on the third day, 14·9 on the fourth day and 21·2 on



the fifth day. In a second period of 6 years, 1897-1902, the mortality was under 5 per cent. of those inoculated on the second day, over 10 per cent. of those on the third day, and about 20 per cent. of those inoculated later.

*The Effects of Antitoxin.*—The membrane ceases to spread, becomes sharply demarcated, separates more readily, is reduced to half on the second day and gone on the third day. The throat swelling and adenitis quickly subside, rhinorrhœa is checked, the toxin is neutralised, and the child becomes more comfortable. The fever subsides, blood pressure rises, and the pulse becomes stronger and less frequent. Complications are rare and less severe.

*Serum Disease.*—The sequels of antitoxin injection are due to the horse serum and not to the antitoxin. They may follow small as well as large doses. The serum acts as a foreign substance and should be used in a highly concentrated form. The serum of some horses is more irritant than that of others. Children are less liable than adults to these sequels and more liable to paralyses. The serum disease begins with a rash in 7-14 days, fever, local or even general adenitis, œdema, occasional joint pains, leucopenia and slight albuminuria. It lasts for 3-4 days. It occurs very frequently and on smaller dosage after re-injections, unless within 10 days. The symptoms, especially local ones, are more severe, and there is intense œdema round the site of injection. The rash comes out within a few hours and may be associated with œdema, rigors, vomiting and collapse (v. Anaphylaxis, p. 905).

Sudden death and fatal collapse have followed antitoxin injection, probably due to the status lymphaticus or to the injection of serum into a vein. The latter may give rise, because of rapid dissemination, to toxic symptoms within a few minutes, viz. rash, rigors, pyrexia, convulsions, vomiting and collapse. Pain at the site of injection, within an hour or two, is relieved by hot fomentations. Local abscess or cellulitis appears on the third to the twenty-first day, and is most liable to follow large injections. It is not always due to lack of care. Profuse sweating in a few hours indicates constitutional reaction to the drug, whereas a dry skin is a bad sign.

Rashes appear in from 30-80 per cent. Many are transient, slight and liable to be overlooked. Primary rashes are usually urticarial, and begin about the wrists and ankles or in the neighbourhood of the injection, becoming generalised in a few days. The rash generally appears on the seventh or eighth day, or the first to the fourteenth day, and lasts 1-10 days. It gives rise to local itching, and rarely fever or malaise. Erythematous rashes may be so marked in diphtheria, apart from antitoxin injection, as to suggest scarlet fever. The antitoxin erythema may start at the site of injection within a few days. Both varieties are more like blotchy erythema than scarlet fever. They may be delayed until the second or third week, and be accompanied by rise of temperature and throat affection, but no pin-hole desquamation. Circinate erythema, the common type of secondary



rash, may follow urticaria after a short interval, or be preceded by a measliiform eruption. From a prognostic point of view the urticarial rash indicates response to the antitoxin; so, too, a secondary rash. The greater such response, the smaller is the liability to cardiac or other paralysis. Petechiæ and hæmatoma sometimes occur at the site of injection.

Joint pains, and pains in the muscles and fascia, especially of the thighs and forearms, may occur in the second or third week and last for a few days. The larger joints are affected and there may be some effusion with fever. Albuminuria is not infrequent and adenitis is fairly common, but they are not due to the antitoxin.

*General treatment* consists of rest, isolation and bed for at least 3 weeks, even in the mildest cases. The horizontal position must be maintained for a fortnight; and longer, if there is irregularity or undue rapidity of the heart or the least evidence of dilatation. The child should not be allowed to sit up for any purpose. Rest preserves the strength and reduces the liability to sudden cardiac failure during convalescence. In very mild cases treated by antitoxin the child may be up in a week, if the pulse and colour are quite good. Young children will require some mechanical restraint to prevent them suddenly jumping up in bed. In cases of nasal diphtheria, intubation and tracheotomy, cylindrical cardboard splints are fixed to the arms to prevent the child infecting the eyes, pulling out the tube or interfering with the bandages.

The *diet* must be nutritious and easily digestible, chiefly milk. It should be given by mouth in small quantities about every 3 hours, for distension of the stomach may cause fatal syncope and must be guarded against even during convalescence. Children are often too well fed at this time. For regurgitation give jellies and thick fluids, and feed slowly. Solid food must not be given if it causes cough. *Nasal feeding* is not often necessary, even after intubation or tracheotomy. It must be used cautiously, if at all, in cardiac and diaphragmatic paralysis. It is indicated by inability to swallow, due to pain, swelling, regurgitation or pharyngeal palsy; coughing due to food getting into the larynx; troublesome regurgitation, continued vomiting, or exhaustion following mouth feeding and the refusal of food. Recourse is had to *rectal feeding* during pharyngeal palsy, and for vomiting, difficulty in passing the nasal tube, epistaxis, fright and struggling against nasal feeding.

Stimulants are generally necessary, except in the mildest cases, and should be distributed over the whole 24 hours, a reserve dose being kept for the early hours of the morning, 2-4 a.m., when vitality is lowest. Brandy, strychnia, nux vomica, ether, ammonia, caffeine and digitalis are all useful, especially the first two. They must be used with caution, if there is fatty degeneration of the heart. The poison of diphtheria appears to be directly antagonistic to strychnia, which can be given in considerable doses. It is



most efficacious subcutaneously. Atropine has been recommended to counteract the effect of the poison on the heart muscle. It paralyses the ends of the vagus, reduces its inhibitory action and often accelerates the heart. Tonics, especially iron and strychnia, are given internally.

*Local treatment* is hardly necessary if antitoxin is given, especially in very young children whom it worries and wearies, and may do more harm than good. Membrane must never be forcibly removed, unless very loose and almost separated. The ordinary local remedies recommended for septic conditions of the throat (p. 234) can be used, choosing by preference liq. sod. chlorinatae, chlorine water, liq. hyd. perchlor., peroxide of hydrogen, and permanganate of potash or zinc. For nasal diphtheria syringe or douche gently with alkaline antiseptic solutions (p. 373). Treat laryngeal diphtheria on the same lines as laryngitis.

*Intubation* or tracheotomy may be imperative. For intubation use a tube as large as can be conveniently inserted, made of hard rubber or vulcanite. Wrap the child in a blanket, with his arms by the side and head extended, and place him on his back with a small pillow under the neck. Insert a gag covered with rubber tubing at the left side of the mouth, do not open it widely, and remove it between the attempts at intubation. The tube is sterilised in a warm saturated solution of boric acid and smeared with a lubricant. It is fixed into the introducer and held in the right hand. The left index finger is hooked round the epiglottis and the tube inserted, along this as a guide, in the median line. The attempts at introduction must be made quickly and very little force is needed. Some operators prefer the sitting posture, with the head held erect and well lifted up to prevent approximation of the epiglottis to the glottis. When the tube is in the pharynx, violent cough is induced but it soon subsides; the respiration is hissing in character, breathing tubular, and the dyspnœa is relieved. It may enter the œsophagus and be swallowed. The general difficulties of insertion are œdema, a thick membrane in the larynx, spasm of the glottis, the end of the tube entering the ventricle of the larynx, and a ring of sub-glottic œdema about the level of the cricoid. Spasm gives way on inspiration, if gentle pressure is kept up. A silk string is fixed to the tube and fastened by plaster in the neighbourhood of the ear, thus enabling the tube to be removed at a moment's notice. The tube is kept in for 24 hours, removed daily for cleaning, kept out a short time at the end of 36 hours, and can generally be dispensed with in 3 or 4 days. It may be rejected on coughing; may increase the dyspnœa by pushing down loose membrane or tenacious mucus, or by becoming slowly or suddenly blocked; or may induce vomiting. If the breathing is not immediately relieved, the tube must be removed. It may give rise to abrasion or ulceration (pressure ulcers) of the mucus membrane, "thread ulcer" due to the silk, may slip into the bronchus or trachea, or cause stenosis of the larynx. If it increases the dyspnœa, do tracheotomy. When no string is attached the tube is



removed by an extractor, or by backward and upward pressure on the trachea below its lower end. On several occasions it has been swallowed and evacuated without difficulty.

The disadvantages of intubation are obvious. It requires considerable experience and affords less complete relief; false membrane is less easily got rid of; and subsequent feeding is more difficult. The patient needs constant skilled attention, while the tube is in position, for at any moment it may be coughed up or have to be removed, and, as it can only be replaced by a skilled person, there is constant risk of sudden asphyxia unless an expert is always at hand. Sudden asphyxia from blockage of the tube may necessitate its immediate removal, and even tracheotomy.

On the other hand it is advantageous in that no anæsthetic is required, and no cutting operation. There is no liability to wound infection, no subsequent scar, less risk of lung infection, and it need not be postponed until imperative. It can be done quickly without assistance and in a bad light. There is less risk of subsequent hoarseness, and recovery is quicker. The voice may remain husky for a few weeks.

After-treatment of intubation consists in careful feeding, usually by the mouth or by nasal tube. If given by mouth, the food should be semi-solid; there is comparatively little risk of any getting into the larynx. The child should be kept on the side, not on the face, to facilitate the escape of mucus, and cough is induced by sips of water every 2-3 hours to clear the tube. Nasal feeding is sometimes needed after the tube has been removed.

*Tracheotomy* is required if the obstruction is rapidly increasing, recession more marked, temperature rising, respiration and pulse rate more frequent, and if the child is more restless and cyanotic. It can be done almost as rapidly as intubation, but it increases the risk of broncho-pneumonia and creates an absorbing surface. If possible, time should be allowed for antitoxin to act. It is advisable to avoid the use of feathers, steam tents and overfeeding. Do not keep the tube in longer than is necessary. Remove it frequently and re-insert if required, and leave it permanently out in 48 hours if possible. The chief difficulty in removal of the tube is that it may be followed by severe dyspnœa, which may come on at once, in a few hours or even during sleep. This is sometimes caused by injury to the cricoid cartilage, interfering with proper action of the crico-arytenoid muscles, spasm of the glottis or granulations of the mucosa. Granulations must be treated by astringents or removal. Sometimes the inability to do without the tube is due to mere nervousness, occasionally to abductor paralysis, and rarely to a ring-like stricture of the trachea. Active interference is of doubtful value. Attempts should be made to remove the tube when obstruction is least marked, and repeated every 3 or 4 weeks. Early removal of the tube, in 48-72 hours, is the best preventive.



*Stenosis of the larynx* may follow either intubation or tracheotomy. It is usually due to granulations but may result from cicatricial contraction, swelling and infiltration of the mucous membrane of the arytenoids, or a chronically inflamed condition of the sub-glottic region. Occasionally there is only a pin-point orifice or practically complete atresia. It is treated by tents and bougies; dilators and intubation tubes; intra-laryngeal division of the stricture; and other surgical measures. Polypoid granulations may occur although tracheotomy has been quickly and easily performed and the tube left in only a few days. They give rise to increasing dyspnoea, after a period of health, and necessitate further operation.

*Treatment of Special Symptoms.*—Paralysis is partially prevented by complete rest at the onset of the disease, recumbency during its course, and rest during convalescence. Antitoxin has occasionally seemed of value but may cause aggravated serum disease, because of anaphylaxis. It is given every 3-5 days. Food must be warm, not hot, liquid or semi-solid, and given in small mouthfuls. Nasal feeding and nutrient enemata may be needed. See that the bladder and bowels are evacuated. The chief measures for curing the neuritis are rest, liberal diet, tonics and moderate stimulation. Galvanism, massage and strychnia can be used, provided there is no acute tenderness. For cardiac and respiratory symptoms try oxygen inhalations, faradism of the vagus and injections of camphorated oil, sparteine or strychnia. Atropine and strychnia are given for pseudo-bulbar crises. In early cardiac palsy avoid drugs likely to cause vomiting, and use enemata for constipation. If the diaphragm is paralysed, causing cyanosis and accumulation of mucus in the pharynx and bronchial tubes, give large doses of belladonna or atropine by mouth and strychnia sub cutem. Adrenalin is probably of no value, unless injected into a vein, and it is doubtfully advisable to raise peripheral blood pressure if there is the least sign of degeneration of cardiac muscle.

For vomiting in early stages, due to deglutition or nervousness, rely on nasal feeding, nutrient enemata, and large doses of belladonna and bromide. In late stages it may be relieved by saline enemata, bromides per rectum and morphia sub cutem. Saline injections relieve thirst, increase diuresis and assist in evacuation of the toxin, reduce restlessness and raise blood pressure. Anuria is relieved by hot packs, and saturated solution of mag. sulph. in drachm doses as a diuretic.

*Convalescence and Prophylaxis.*—Gradually the child is allowed to sit up in bed by increasing the number of supporting pillows, and so he progresses to the use of a bed rest, to sitting up in bed without support, on a sofa, walking about the room and then out-of-doors. Convalescence is greatly aided by fresh air, so in suitable weather the bed may be placed near an open window. Tonics, notably iron preparations, are necessary.

The period of *quarantine* is an open question. If no bacteriological examination is made, the child should be isolated for 3 weeks after the



membrane has gone. Probably in most cases there is no infectivity a few days after, and the child may be allowed to go to school in another week. It is safer to insist on 6 weeks quarantine. Swabs should yield no specific bacilli on culture, but they have been recovered from the throat even on the 363rd day, though they are not likely to be virulent as late as this. The usual methods of isolation and disinfection of all utensils, discharges, etc., are necessary. Antiseptic treatment of the nose and throat reduces the infectivity. These structures should be examined bacteriologically in all "contacts" and, in schools, children in whom the result is positive or doubtful should be isolated and treated with hydrogen peroxide spray or gargle. A "contact" can spread the disease without developing it. The injection of 200-500 units of antitoxin every 3 weeks has been recommended, but is not advisable on account of the risks of anaphylaxis and the possibility of having to treat an actual attack of the disease.

*Anaphylaxis.*—This is a curious constitutional state set up in a patient by the injection of antitoxin, rendering him liable to serious and even fatal symptoms if a further injection is given *after* an interval of 10 days, say within another 2-3 weeks. It is due to the protein substance in the serum and has been known to last more than a year. A similar condition follows the injection of milk and egg albumin. The symptoms are those of aggravated serum disease. It is an important peculiarity, for it makes it essential to exercise great care in the repeated administration of the drug. If given within 10 days the effect is not produced.



## CHAPTER LXIV.

### INFLUENZA.

Influenza is an acute specific infective febrile disorder of variable severity and extremely difficult of diagnosis in its milder manifestations. Children of all ages are liable to it, less so than adults, but they are comparatively immune from its complications and dangers. Attacks are generally mild and followed by slight catarrh and profuse sweating. Cases have been recorded on the first and second days of life, possibly of congenital origin, and occur occasionally in the early months. A certain amount of immunity is conveyed to breast-fed infants.

**Pathology.**—It is due to Pfeiffer's bacillus. This organism is not invariably present and has been found in other diseases. It resembles the bacillus found by Sprengel and Eppendorf in pertussis and the Koch-Weeks bacillus, and is readily stained by carbol-fuchsin and Canon's dye. Many attacks of so-called influenza are due to organisms belonging to the same group but differing in slight morphological and cultural details. The organism has been found in the nasal secretion, sputa, cerebrospinal fluid, pus from the ears, etc., and occasionally in the blood. It probably enters through the nose or naso-pharynx. Its effects are mainly due to a toxin which acts primarily on the nervous system. In some cases the chief effects fall on the gastro-intestinal, respiratory or blood-forming organs.

**Symptoms.**—The incubation period is uncertain, probably 1-3 days. The onset is variable, and often sudden with fever, headache, general malaise and pains. Lassitude and slight fever may be the only signs. Sometimes it is ushered in by epistaxis, sore-throat, coryza or gastro-intestinal symptoms. Furred tongue, loss of taste, anorexia, flatulence, nausea, vomiting and constipation are common symptoms. Occasionally an evanescent erythema is noted. The pulse is unduly rapid, rarely slow and irregular. Cases may be grouped according to the predominant symptoms. In practice it is common to find various combinations of the different symptoms but sometimes the attack can be definitely labelled as belonging to one or other group. The *febrile type* is frequent in children. The temperature is raised for a day or two, rising suddenly, perhaps to 105° F., and subsiding rapidly or coming down gradually by lysis in the course of a week. Such a temperature is common in every kind of attack. The child is ailing and languid, with loss of appetite, and sweats readily.



The *gastro-intestinal type* is also common in children, and liable to be misconstrued for it simulates acute febrile gastro-intestinal catarrh. The symptoms are marked colic, restlessness, constipation and moderate fever; or vomiting and convulsions, suggestive of the onset of tuberculous meningitis. In other instances there are loss of appetite, furred tongue, vomiting and constipation, followed by diarrhœa in a few days. Or there may be much diarrhœa and abdominal pain, sometimes hæmatemesis and melæna. The spleen may be enlarged.

The *catarrhal type* may be naso-pharyngeal in origin or simulate the onset of measles. Retro-pharyngitis is common at the onset and a local fibrinous exudation forms later. Sometimes there is a tonsillitis which may be membranous; or pronounced coryza and epistaxis; or coryza, conjunctivitis and cough. Earache is often present.

In the *pulmonary type* the air passages are rapidly affected. Inspiratory crepitations are heard throughout the lungs, especially over the posterior parts. There is no dulness. Cough is dry, paroxysmal and difficult. This œdematous state of the lungs is apt to wander from one part to another, and is followed by profuse muco-purulent catarrh.

*The nervous type.*—Infants may exhibit a marked somnolence, due to the toxæmia and lasting for even 7-10 days. It may be associated with high fever. It is not infrequent in older children, but in these the more common nervous symptoms are headache, sleeplessness, severe depression, aching bones, sensitive spine, general pains, and perhaps mental aberration and various forms of neuritis and palsy. Meningeal irritation is easily set up, and possibly an influenzal meningitis or encephalitis may occur. Retraction of the head, irritability and fever are usually toxic and of a few days' duration. More prolonged cases are inflammatory, due to the specific bacillus or secondary infection, and may end fatally. Occasionally cerebral and peritoneal irritation are combined. Thus, an infant developed a pseudo-peritonitic attack and well marked symptoms of basal meningitis after influenza, but recovered in a few days. Another boy, aged 7 years, was dazed and listless on the eighth day, had abdominal pain on the eleventh, and paroxysmal pain with inability to pass water and constipation two days later. He was drowsy with dilated pupils, furred tongue, and retention. On the sixteenth day he was noisy and presented general hyperæsthesia, pain in the back of the neck and slight retraction of the head. On the following day he was irritable and complained of pins and needles down the legs and pains in the back as of "fleas biting him." After this he steadily recovered. There was no fever throughout, except possibly at the onset. In another case there was considerable muscular, nervous and psychical derangement one month after influenza. The girl,  $2\frac{3}{4}$  years old, was unable to hold up her head, weak on her legs, and fell on attempting to walk or exhibited a gait like cerebellar ataxia. She moaned all day, made faces, did not talk, was not interested in toys or



games, and kept picking her face and throwing the head and trunk about. She was strange in her manner and had got very spiteful, biting the other children if she had the chance. Twitching of the face, eyes and hands was present during sleep. The pulse rate was 120 and the knee jerks were exaggerated. Recovery was complete.

Occasionally the toxin acts powerfully on the heart, causing tachycardia, arrhythmia, attacks of collapse with small thready pulse, or even angina. These are apt to occur even under 1 year of age and may be regarded as complications. Asthenia and general hyperæsthesia are sometimes very marked. Headache is usually frontal and may be temporal. It is often severe in older children and slight or absent in infants.

**Complications** are mainly those of the pulmonary, cardiac and nervous systems. They occur at the onset, during the course, or after the fever. Attacks of syncope, anginal in character, are not infrequent. The child becomes very pale, cold and almost pulseless, or the pulse is frequent, small and thready. Similar attacks may follow the old-fashioned influenza cold. Tachycardia and arrhythmia are common at any period of the disease. At the onset the pulse is unduly rapid in proportion to the fever, just as in scarlet fever. The cardiac symptoms are due to the action of the toxin on the cardiac muscle or nerves, or secondary to neuritis of the cardiac nerves. Parenchymatous degeneration may be found after death. Sometimes there is dilatation of the heart, with cyanosis and collapse.

Catarrh of the air passages is part of the pulmonary type of the disease. There may be a wandering patchy œdema of the lungs, general capillary bronchitis, or broncho-pneumonia. In rare instances the pneumonic affection is insidious and atypical, creeping about the lungs and in time affecting practically the whole of them, one portion becoming involved as another recovers. Pneumonia is usually lobular, if due to the specific bacillus, and often not recognised as influenzal. Each kind of pneumonia may be primary or secondary to pneumococcal infection. Pleurisy is not uncommon, lobar pneumonia may occur during convalescence, and asthma is an occasional sequel.

Convulsions are not infrequent in babies and may recur for several days. In the early stages headache, neuralgic pains, delirium, somnolence and coma are due to the action of toxin on the central nervous system. Later on, toxic or inflammatory affections may develop, e.g. meningitis of the brain or cord, encephalitis, neuritis, degenerative changes in the nerves analogous to those in diphtheria, myalgias, and neuralgias of various kinds, functional or organic. Mental disturbances, varying from simple fatuity to acute mania and insanity, may occur at any period but most often in convalescence. At this period hallucinations, lethargy, stupor and loss of memory are not uncommon. Sometimes epilepsy, hysteria and neurasthenia date from an attack of cerebral type. The cranial nerves are



apt to suffer, especially the sixth and third nerves. Profuse sweating is a frequent nervous symptom.

Otitis media (p. 995) is common and the organism may be found in the pus. It is unilateral in 75 per cent. and sometimes followed by mastoiditis. Albuminuria is present in 5-10 per cent., and acute glomerulo-nephritis is an occasional complication. Retention is due to paresis of the bladder and secondary pyelitis may ensue. Dudgeon and Adams (1907) reported a case of pyæmia, multiple arthritis and meningitis in a 10-year old girl. The primary lesion was an epiphysitis of the upper end of the radius.

**Diagnosis.**—Much importance must be attached to a history of exposure to infection and the presence of other cases in the house. At the onset the cough may suggest measles and later on be like that of pertussis. The diagnosis from the simple influenzal cold depends on bacteriological examination and is relatively unimportant, for the same treatment is necessary. Cases ushered in with erythema, rapid pulse and vomiting, are difficult to diagnose from scarlet fever, if there is any sore-throat. The pulmonary type may simulate incipient phthisis. Gastro-intestinal attacks are like (1) acute appendicitis, perforated gastric ulcer or intestinal obstruction; (2) typhoid fever, acute enteritis or colitis; (3) Henoch's purpura. The nervous type simulates meningitis or encephalitis, especially if associated with an infrequent irregular pulse, somnolence, and cranial nerve palsy. But it ends in recovery, though the squint may take 3 months to disappear.

**Prognosis.**—Death rarely occurs in the young, except from cardiac failure or secondary pneumonia. The outlook is good unless the heart is affected. In babies it must be guarded, for sudden failure may develop at any period within a week of the onset, especially if the pulse is unduly rapid or irregular. Usually recovery takes place in a few days, but some uncomplicated cases last for weeks. Possibly the infection is a cause of prolonged anomalous fever. Convalescence is slow and marked by lack of appetite, debility, sweating, cough, fractiousness, cardiac weakness and liability to secondary infections. There is no special tendency to tuberculosis.

**Treatment.**—Isolate the child and give a mixture of ammoniated quinine, acetate of ammonia, and syrup of orange. Keep the patient in bed, and avoid cold and exposure. Order a reduced diet and plenty of fluid in the febrile stage, but encourage the appetite and feed the patient as soon as possible. A dose of calomel or castor oil is beneficial. In the early stages give phenacetin as a sedative, antipyretic, and for the relief of headache. Phenacetin gr.  $\frac{1}{2}$ , caffein citrate gr.  $\frac{1}{4}$  and sod. bicarb. gr. 1 can be given every 2 hours at 1 year of age, and in double quantities at 3-4 years, for 6-8 doses. For older children aspirin or salicylate of soda can be used instead of the bicarbonate. At all ages aspirin may be given alone in small frequent doses. The heart must be carefully watched and



stimulated with strychnia, caffeine, strophanthus or digitalis, if necessary. Euquinine, quinine and salicylate of quinine are also useful. Amyl nitrite has been advocated for profuse sweating. For high fever and delirium apply ice to the head and give tepid baths. Other treatment is symptomatic. Iron, arsenic and cod-liver oil, with change of air, are needed in convalescence. Special care is necessary because of the liability to dilatation of the heart and inflammation of the lungs.



## CHAPTER LXV.

### TYPHOID FEVER—PARATYPHOID FEVER.

Typhoid fever is a general blood infection, via the intestinal mucosa except in intra-uterine life when the infective organism is conveyed to the foetus by the umbilical vein. The chief characteristic of the disease in early life is its comparative mildness, though at times it is just as severe as in adults. On account of this peculiarity it is probable that many cases in infants, especially in hospital practice, are overlooked. Abortive attacks are frequent and the intestinal symptoms not severe. Vomiting and head retraction are more common, hæmorrhage and perforation infrequent, and diarrhœa and abdominal distension less marked than in later life. The babe suffers from a febrile attack and diarrhœa, is supposed to have enteritis or entero-colitis, and gets well. Typhoid fever may not even be suspected and, if it is, the difficulty of diagnosis is great. In older children the attack is often of the ambulatory type. It is more common in boys than girls, probably by reason of their habits at games, such as marbles, whip top, etc., rendering them more liable to infection, if playing in the vicinity of ash-pits. It is uncommon before the fifth year of life. In breast-fed infants the infection is conveyed by water used in bathing, directly from the mother, or through the agency of flies.

**Congenital Typhoid.**—Typhoid fever in the mother leads to abortion in about two-thirds, generally in the fifth month of pregnancy. In 10 out of 21 cases collected by Hicks and French (1905) the bacillus was transmitted to the foetus, delivery usually taking place in late stages of the fever and the child sometimes being born alive. In these cases bacilli were generally present in the foetus, whereas in cases of abortion early in the course of the fever bacilli were not found. This is an important point for it suggests the advisability of inducing labour early in the fever, if the child is viable, in order to prevent intra-uterine infection. Though the child may be born alive in late stages of the fever, it almost always succumbs. Newborns and very young infants of women with typhoid may exhibit Widal's reaction but no symptoms of the disease. Such babes have had the fever in utero or have acquired the agglutinating principle from the maternal blood or milk. On the other hand the serum reaction is often absent in the foetus. Like other fevers, typhoid in the mother may cause malnutrition or pathological metabolic and organic changes in the foetus through the transmission of toxins. Such infants may be physically or intellectually defective.



*Symptoms.*—Diarrhœa, fever and splenic enlargement are the most frequent signs in those born alive. Occasional symptoms are rose spots, slight jaundice, hæmorrhage, tympanites, vomiting, constipation, convulsions, cough and purpura. Hæmorrhages may take place into the skin, liver and kidneys. The mesenteric glands are a little enlarged. Intestinal lesions may be absent or resemble those of ileo-colitis. Definite ulceration of Peyer's glands has been noted. According to Helwig Peyer's glands are fully developed by the sixth month of foetal life. In Blumer's case (1900), a full-time child died from hæmorrhagic disease, the mother having had typhoid fever 4 months before confinement, and the specific organism was found.

**Infantile Typhoid.**—The symptoms during the first 30 months of life are well shown in a summary based on nearly 400 cases collected by J. P. Crozer Griffith and M. Ostheimer (1902).

					First Year.	Second Year.	Age 24-30 months.
<i>Number of Cases</i>					139	187	68
Fever	..	..	..	..	46	50	42
Diarrhœa	..	..	..	..	42	40	24
Rash	..	..	..	..	26	27	23
Spleen	..	..	..	..	26	26	13
Tympanites	..	..	..	..	18	18	12
Vomiting	..	..	..	..	11	9	8
Constipation	..	..	..	..	1	6	3
Hæmorrhage	..	..	..	..	1	1	1
Perforation	..	..	..	..	0	2	0
Cough	..	..	..	..	1	20	6
Fits	..	..	..	..	4	6	2

Other symptoms in the first year were collapse 3, jaundice 3, and otitis, albuminuria and noma 1 each; in the second year, collapse 1, epistaxis 4, laryngeal obstruction 2, otitis, meningeal signs, aphasia, parotitis and desquamation 1 each, and relapses 6; in the third period, collapse 2, epistaxis 1, meningeal signs 3, pneumonia 3, otitis 1, phlegmasia alba dolens 1, and relapses 3.

In comparison with adult typhoid it is noticeable that diarrhœa is almost invariably present, though occasionally constipation may be intense throughout; vomiting is more frequent; tympanites is rarely severe and sometimes the abdomen is retracted; and epistaxis in early stages is infrequent. Many cases present the symptoms of enteritis, viz. green and watery stools, slight or absent tympanites, irregular fever, cerebral irritation, rapid emaciation, prostration, albuminuria and weak heart sounds. The duration is 1-8 weeks; more than two-thirds of the cases lasting 1-3 weeks, and a few less than 1 week.



**In Older Children.**—Both in infants and children the onset is generally indefinite. Feebleness and somnolence may mark a prodromal stage of 2-14 days' duration. It is sometimes ushered in with vomiting and severe abdominal pains; or with vomiting and high fever, up to 104° F. in 48 hours, rarely a rigor; and may simulate meningitis. In children the common early symptoms are anorexia and perhaps restlessness, headache, drowsiness at unusual times, moderate vomiting and constipation. The onset is generally more rapid and less insidious than in adults. Epistaxis is present in less than half the cases.

The *fever* rises by steps to its maximum in 3-5 days and is high but well tolerated. It may last for several days and the child scarcely feel ill. Its average duration is 2-3 weeks. In one case under my notice the temperature chart was characteristic but the fever only lasted for 10 days. The girl, 10 years old, had no definite symptoms. She was treated for typhoid during the fever and a period of 10 days apyrexia. A typical relapse then occurred, with a copious rash on the thirtieth day of the illness. The fever persisted for 8 months and terminated in death from marasmus, though there were no definite symptoms throughout.

In several other children the fever only lasted 10 days. Usually it lasts 2-3 weeks and falls by lysis during a period of 7-10 days, sometimes more quickly, and occasionally by crisis. The pulse rate corresponds with the height of the fever or is lower. It is seldom dicrotic.

*Rash* is common, at times abundant, and may appear early, generally in 7-10 days. The number of spots is no measure of the severity of the disease but duskiness is a grave sign.

*Anorexia* is marked. The tongue may remain almost clean. Usually it presents a thick white coating, dark border and clean tip, forming the "typhoid triangle." It is dry, brown or black, and cracked in severe cases. Sordes on the teeth, rhagades and foul breath are then noted. Vomiting is not infrequent at the onset, throughout the course, and as a terminal symptom. Tenderness and pain in the abdomen may be present. Diarrhoea is about as frequent as constipation. It may appear late, is rarely severe, but is sometimes grave. Tympanites is fairly common after the first week unless the diet is very carefully regulated. Intestinal hæmorrhage and perforation are uncommon in the very young because of the infrequency of severe ulceration.

The *spleen* is almost invariably enlarged, often very early in the disease. In a 10-year old boy, taken acutely ill with no definite symptoms, it was so large and hard that a diagnosis of malarial fever had been given.

The *nervous symptoms* sometimes predominate over the intestinal ones, but are rarely severe, especially in the younger children. They take the form of restlessness, insomnia, fatuous stupidity, extreme nervousness and fear, paroxysms of terror, or intermittent and perhaps violent delirium.



Headache, drowsiness, dulness and apathy are quite frequent. The typhoid state is rare.

*Blood Examination* shows no initial leucocytosis. Leucopenia is progressive after the first and up to the fifth week. The percentage of hæmoglobin and eosinophiles is reduced. The bacillus is often present, even in early stages, and may be found in film preparations. It gets into the blood, via the lymph glands and spleen, and it is doubtful whether it multiplies in the blood. The disease is a true septicæmia. W. Coleman and R. H. Buxton found the organism in the blood in 75 per cent. of 123 cases. Ten c.c. of blood were taken and ox gall, peptone and peptone glycerine were used as culture media. The usual method of culture is to receive 5 c.c. of blood into 300 c.c. broth and incubate it. This is hardly practicable, and rarely necessary, in private practice among children. The bacillus has been found in the blood during the incubation period (Conradi), and after 6 weeks' freedom from fever (Gennari and Lesieur).

*Widal's Test.*—The agglutinating power of the serum is weaker in children than adults, appears earlier, and does not persist as long. It can be transmitted through the placenta and maternal milk but is then of comparatively short duration. The reaction may persist for 10 years, usually less than 5 years, after recovery. It is, therefore, sometimes present in cases which are not typhoid. Complete clumpings should occur within half an hour with a dilution of 1 in 200. Negative results suggest a wrong diagnosis, though there are genuine cases in which the reaction is absent. It may not appear until late in the disease or not until convalescence. It is generally present in the second week, rarely as soon as the fifth day, and may be absent throughout. On the whole it is a valuable confirmatory sign and perhaps the first positive one. Thursfield (1901) obtained the reaction in 42 out of 100 sick children who presented symptoms more like typhoid than anything else.

The *urine* often contains albumin, sometimes casts and renal epithelium, and is occasionally nephritic in type (renal typhoid). Ehrlich's diazo-reaction, due to the presence of some aromatic body, is almost always present in the second week and before the Widal test is positive.

**Morbid Anatomy.**—The anatomical lesions are much less marked than in adults. There is a catarrhal ileo-colitis or enteritis, with infiltration of Peyer's patches and the solitary follicles, enlargement of the mesenteric glands, and a large, soft spleen. Ulceration is uncommon in the first year of life. In older children it may be very extensive and involve the cæcum and large intestine. Swelling of the agminated and solitary glands in children is not distinctive of typhoid, for it is also found in the status lymphaticus and may be produced by various toxic processes. On the other hand absence of such swelling is not conclusively negative, for some such cases give Widal's reaction and run the course of typhoid. Degeneration



of the heart, liver and kidneys, bronchitis and broncho-pneumonia, and hyperæmia of the meninges and brain may be present.

**Complications and Sequels** are comparatively infrequent. *Haemorrhage* occurs in 1-5 per cent. It is more common than perforation but is rare under 5 years of age. Sometimes there is little constitutional disturbance. Serious bleeding causes sudden fall in temperature and rise in pulse rate; perhaps collapse, pallor, pinched nose, cold sweat and cold extremities. Blood is passed per anum in a few hours or next day.

*Perforation* is found after death in many cases in which it was not suspected during life. It is more frequent in girls than boys, but rare in children, e.g. 4 out of 210 (W. J. Butler, 1905), 9 out of 375 (Kissel, 1904), 0 out of 277 (Wolberg). It is most frequent in the third week, sometimes as early as the tenth day, or not until the fifth week and later, or during a relapse. It occurs in both mild and severe attacks. The site is in the ileum 80-85, large intestine 10-15, and appendix 5 per cent., occasionally in the jejunum or a Meckel's diverticulum. Its onset is gradual or sudden, and the symptoms often obscure, latent or absent. Attention should be paid to the state of the abdomen, pain, pulse-rate, condition of the patient and the blood count.

Warning symptoms are abdominal pain and tenderness, local or general; rising leucocytosis; local muscular rigidity and tenderness; and hæmorrhage. Some cases are ushered in with rigor, pain and rise of temperature; others with severe vomiting and hiccough. The pain is sudden, intense and paroxysmal; diffuse or localised; in any locality, especially the right iliac fossa; and may radiate to the pubes or penis. It may pass off in a few minutes, last for days, be associated with vomiting, or be absent altogether. Local tenderness is usually present.

The abdomen is flat or distended, rigid, moves on respiration or is perhaps fixed, and is tender. Liver dulness is abolished. Tympanites is due to peritonitis and often associated with vomiting. The temperature falls suddenly from collapse or peritonitis; it may rise. The pulse and breathing suddenly increase in frequency, and respiration is shallow and costal. Hiccough, vomiting, sweating and shivering are sometimes present. The child is restless, looks ill and anxious, and exhibits symptoms of collapse. Secondary signs of peritonitis develop. Leucocytosis is usually present early. Sudden abdominal pain and tenderness, with leucocytosis, warrant operation. Leucocytosis is not positive proof, and may not be marked until peritonitis and collapse have supervened. Moreover, the signs of perforation and leucocytosis may be present, although the gut is intact. There may be leucopenia.

The diagnosis is difficult and the prognosis bad. Abdominal pain in the course of typhoid fever is due to other causes, viz. gastritis, colic, constipation, fæcal impaction, ileo-colitis, diarrhœa, distended bladder,



intestinal strangulation, rupture of an infected mesenteric gland; peritonitis, appendicitis, cholecystitis, iliac phlebitis; pleurisy or pneumonia; and hæmorrhage or, indeed, no definite cause. The prognosis is most favourable if the perforation is in the appendix. About 90 per cent. die, one-half within 2 days, unless treated surgically. No opium is permissible until a diagnosis has been made and the question of operation decided. Exploration can be done under local anæsthesia if necessary. A general anæsthetic is preferable. Many cases can be saved by early and rapid operation, 30-40 per cent. according to Osler. The first operation was done by Kussmaul in 1887. Finney (1889) collected 112 cases and Keen added 38 a few months later. Out of 111 no less than 27 recovered; 6 out of 12 under 15 years of age. A girl under my care recovered but was left with a fæcal fistula which slowly healed. Operation for secondary peritonitis almost invariably fails to effect a cure.

The *nervous symptoms* are numerous. Sometimes there are signs of meningeal irritation (meningismus) though nothing abnormal is found after death; or serous meningitis, with bacilli in the cerebrospinal fluid, and œdema and round-celled infiltration about the blood vessels; or a purulent meningitis. The clinical signs are of varied type (Rocaz and Carles, 1907):—(1) Intense headache, frequent vomiting, constipation, irregular pulse and breathing, hyperæsthesia, vasomotor disturbance and head retraction. (2) Signs like those of cerebrospinal meningitis, viz. Kernig's sign, rigidity of neck, spine and limbs, and symptoms as in the first type. (3) Convulsions, rapidly fatal, in infants. (4) Clinically like tuberculous meningitis. The fluid obtained by lumbar puncture contains in the first type pus and typhoid bacilli in pure or mixed culture; in the second, turbid or transparent fluid with typhoid or other organisms; in the third, normal fluid under high pressure; and in the fourth, clear fluid and abundant lymphocytes. The last 2 varieties are apparently the result of the action of toxin on the meninges. Lumbar puncture is essential for diagnosis, for the removal of organisms and toxins, and for the reduction of pressure.

Ocular palsy, aphasia, spastic hemiplegia, Jacksonian epilepsy, tetany and mild peripheral neuritis have occurred. The *psychical changes* are mania, dementia and melancholia in order of frequency, or a more or less confusional state. Marked dementia is the most grave. Most of these patients get well.

Other complications and sequels are bed-sores, due to emaciation and mal-assimilation, and not always preventable; boils, abscess, hæmorrhagic bullæ, purpura; pneumonia, pleurisy, empyema, gangrene of lung; perichondritis of the thyroid cartilage, in a 12-year old boy, which necessitated tracheotomy; stenosis of the larynx, secondary to perichondritis; necrosis of the jaw; myositis of the rectus abdominis; cholecystitis, perforation of the gall bladder; general septic peritonitis, without per-



foration; arteritis, with or without thrombosis, and perhaps gangrene; otitis media, secondary parotitis, noma, recto-vaginal fistula, purulent arthritis, and necrosis of bone. Myocardial degeneration is dangerous and apt to be overlooked. It is more chronic than in diphtheria, but may be acute and severe. In one girl tuberculosis developed during the fever.

**Course and Prognosis.**—The disease is milder and more variable than in adults. Some cases are abortive, afebrile, ambulatory, or mild and lasting only 8-10 days. Others are as severe as in adults. In renal typhoid pyrexia and hæmaturia are the only symptoms, but the bacilli are found in the urine and Widal's test is positive. A pneumonic type, like broncho-pneumonia at the onset, is rare. Anorexia, fever, diarrhoea and restlessness may lead to profound emaciation. The hair may fall out, and the nails show transverse furrows or drop off. The temperature is variable and may fall by crisis. Branny desquamation is not uncommon, and even large flakes may be cast off. It begins at the end of the febrile period, in 10-15 days, in the axilla and spreads to the trunk and limbs but not to the hands or feet. Bad signs are severe meteorism, weak cardiac first sound at the apex, thready pulse, severe diarrhoea, picking at the bed-clothes, waking delirium, foul mouth and insensibility. Sudden death is due to toxic action on the vagus, reflex abdominal irritation, perforation, collapse, or myocardial degeneration. It usually results from syncope in the third week and has occurred in latent and unsuspected cases. The mortality is small, about 3-5 per cent., and lower during the first than the second 5 years of life. Green vomiting and diarrhoea are very unfavourable. A high pulse rate and low temperature may indicate hæmorrhage. Anæmia and debility may persist for months, and there is frequently a tendency to obesity in convalescence.

**Relapses.**—Relapses develop in about 15 per cent. The younger the child, the more likely are they to occur, and there may be more than one. Persistent splenic enlargement is presumptive evidence in favour of relapse. The temperature may rise by steps. More frequently it rises rapidly, keeps high for a time, and falls quickly. Mild abdominal symptoms are present in about half, and a rash in three-fourths of the cases. Complications are mild and infrequent. A relapse lasts 1-2 weeks, possibly 5 weeks, and is rarely fatal. Succeeding relapses are milder. In exceptional cases intercurrent relapses occur, the temperature rising again before the fever of the preceding relapse has subsided. These are occasionally serious and prolonged cases, and likely to prove fatal from asthenia.

**Diagnosis.**—Green stools, tympanites, listlessness and mild pyrexia cannot be distinguished from enteritis except by Widal's test, the diazo-reaction, and the blood count. Gaertner infection is diagnosed by agglutination tests and the recovery of the bacillus from the stools. The diagnosis of paratyphoid depends on the serum reactions. At the onset the disease is liable to be mistaken for enteritis, appendicitis, peritonitis, lobar



pneumonia with delayed physical signs, malaria, meningitis and miliary tuberculosis. Much stress can be laid on the character of the temperature chart in the first few days and on splenic enlargement, constipation and rose spots.

**Treatment.**—Prevention of the spread of the disease consists in anti-typhoid inoculation of those exposed to infection, and *disinfection* of the excreta of those infected. Inoculation is practically never required for children. All clothing and bedding should be disinfected by live steam if available, or by immediate soaking in carbolic acid solution and subsequent boiling. The buttocks must be washed with bichloride of mercury, 1 in 1000. The *stools* contain the bacillus at the beginning of the second week. Add to each two volumes of carbolic acid, 5 per cent. solution, and let them stand for several hours. The Local Government Board recommends corrosive sublimate oz.  $\frac{1}{2}$ , crude hydrochloric acid oz. 1, anilin blue gr. 2, water 3 gallons (= 1 in 1000); it is poisonous and not deodorant. Izal or cyllin 2-5 per cent., and formaldehyde 4-5 per cent. strength are also used. Fresh chlorinated lime, 1 part to 2 of water, is satisfactory. The dejecta must be covered with it. Air-slaked lime is valueless. It should be freshly burnt and mixed with water. Use freshly made milk of lime in the proportion of 3 volumes to 1 of fæces. Add 60 parts of water slowly to 100 of lime. Milk of lime is made from this by mixing 1 part by weight with 8 of water. It is mixed thoroughly with the fæces by stirring and allowed to stand for 24 hours. The *urine* contains the bacillus in 20-35 per cent. It is turbid and shimmers. It can be disinfected in  $\frac{1}{2}$ -1 hour by formalin 10 per cent., 1 part to 10; carbolic acid 5 per cent., 3 parts to 10; or bichloride of mercury 1 in 1000, 1 part to 40 of urine. A jar containing 750 c.c. carbolic acid or 100 c.c. mercury solution is enough for 1500 c.c. urine. The urine should be poured in and the jar emptied daily. The urinal must be kept in a separate jar of the disinfectant. Chlorinated lime and liquid chlorine are also good disinfectants.

*Specific treatment* is at present only on trial. Chantemesse's serum is sometimes used.

**Diet.**—Impress on the parents that the child cannot digest much food and will emaciate, but will regain weight afterwards. In very acute and severe stages it may be advisable to give water only for 12-24 hours; especially if there is abdominal distension, toxæmia, and the passage of undigested stools. Weak cold tea is harmless, stimulating and checks vomiting. The diet must be such as is suitable for any ulcerative, intestinal condition. It should be easily digestible and assimilable, non-fermentative, leave no solid residue, and contain sufficient nutriment, salts and water. Feed infants on human milk or whey. If the child is hungry and can digest more, give peptonised milk, malted milk, diluted milk, koumiss, butter-milk, dextrinised gruels, meat juice, chicken or veal tea, various broths, Mellin's or Benger's food, liquid custard, beaten up egg, custard



pudding, junket and similar articles. Liquid custard is made of 5 yolks of egg to milk 1 pint, sugar and flavouring agents. Food should be liquid or merely thickened, and must depend in quality and quantity on the character of the stools which require careful examination for curds, fæculent debris, blood and sloughs. If there are curds the milk must be peptonised, diluted or omitted. The caloric value of various foods should be taken into account, e.g. a pint of milk = 450 calories, an ounce of maltine = 240 calories. Other suitable additions to the diet are fruit juice, lime juice, glucose dr. 1 in beef tea  $\frac{1}{2}$  pint, maltose, lactose, treacle, honey, biscuit powder, bread jelly, bread crumbs, cocoa, scraped meat or raw meat pulp.

It is unnecessary to keep the child on a minimum diet, if more can be digested. In fact it is probable that starvation and malnutrition predispose to extensive ulceration, hæmorrhage and perforation. The appetite, the state of the tongue and abdomen, and the character of the stools are the best guides to the amount of food permissible. Although in many cases a liberal diet is beneficial, even during the febrile stage, it is important that it be given in a fluid or semi-solid form. Irritant particles of food are apt to be injurious and much too little regard is often paid to the effects of milk curds. Milk alone may cause diarrhœa.

During convalescence the diet is augmented by increasing the quantity and variety of the articles mentioned. At the end of a week of apyrexia junket, bread sauce and baked custard are suitable additions to a more rigidly liquid diet. In another 3 days a lightly boiled egg and bread and butter are given. After a further 24-48 hours the diet can be rapidly increased to full diet at the end of 14 days freedom from fever. Hunger is often great during the convalescent stages and over-eating must be guarded against.

For *vomiting* rely on diet, bismuth, sod. bicarb. or hydrochloric acid. For *tympanites* give water freely and omit food temporarily; prescribe salol, chlorine water, calomel, or sod. sulphat. in hot water until the bowels act. An ice bag to the abdomen may be used with discretion. For *constipation* regulate the diet by diluting milk, and adding meat preparations, maltose and malted foods, and give enemata or small frequent doses of calomel or sod. sulph. For *diarrhœa* regulate the diet, and give ol. ricini in small doses, bismuth, tr. ferri perchlor., tannigen, tannalbin, and enemata of starch with or without opium. For *hæmorrhage* starve the child and give gelatine, salines sub cutem, and codeia or codeine phosphate; for *perforation*, operate or rely on starvation and opium.

*Antipyretic measures* need rarely be energetic. Cold baths, 10° F. below the temperature of the patient, are unsuitable for profuse hæmorrhage, perforation, and for young children. Rely on an ice cap, cold or tepid sponging, cold compresses and cold packs. Measures which are disliked, or cause excitement and collapse, are apt to do more harm than good. Cold is beneficial as an antipyretic. It relieves bronchitis, delirium and coma;



reduces the pulse rate and temperature ; but does not avert hæmorrhage and perforation, or shorten the disease. The feet must be kept warm. A bath at 95-100° F., with cold to the head, is useful for restlessness and delirium. Quinine is the most suitable drug.

*Intestinal antiseptics* are unreliable in action and irrational measures in practice. Calomel is the best for it reaches the lower part of the small intestine before being converted into perchloride (P. W. Latham). Give it in doses of gr.  $\frac{1}{8}$ - $\frac{1}{6}$  every 4 hours. It is liable to cause stomatitis, ulceration of the gums and diarrhœa. Benzo-naphthol is harmless. Syr. Cyllini may be given, and other drugs already described (p. 300). Liq. hyd. perchlor. and tr. fer. perchlor. are sometimes given in combination for diarrhœa. The latter drug readily parts with its chlorine. Other appropriate drugs are quinine and nascent chlorine ; sulphurous acid with syr. limon. and aq. chlorof. ; ol. terebinth. in small frequent doses ; essential oil of cinnamon, small frequent doses in gelatine capsules or emulsion ; and urotropin for bacilluria. As a general rule the simpler the treatment, the better the results in children. Some of the above remedies are unpleasant to take and apt to upset the digestion. An effervescent mixture of quinine in highly febrile cases, with calomel in small doses if there is constipation, gives good results. Opium is rarely needed. A drop or two of the tincture may be given, if the nervous symptoms are prominent and mental rest requisite. It is often inadvisable, for the symptoms depend on toxæmia and elimination is of most importance.

*Alcohol* is frequently required. It must be given if the first sound of the heart fails and is useful in high fever. Brandy is the best stimulant. For *cardiac failure* give also digitalis, strychnia, nux vomica, caffeine and sal volatile ; for *toxæmia*, high enemata of saline solution to stimulate renal excretion ; for *collapse*, camphorated oil sub cutem, or tr. capsici m. 5-15 in oil or water per rectum (Ager, 1906), repeated every 10-15 minutes for 2 hours if necessary, in addition to other remedies ; and for *headache*, restlessness and fever rely on hydrotherapy. An occasional small dose of phenacetin and caffeine is permissible.

### PARATYPHOID FEVER.

There are many organisms intermediate between the colon and typhoid bacillus. Apparently b. coli, paracolon bacillus, paratyphoid  $\alpha$ , paratyphoid  $\beta$ , and the typhoid bacillus represent a series of a group, to which also belong b. enteritidis of Gärtner and b. psittacosus. The paratyphoid  $\alpha$  is culturally like the paracolon bacillus. The paratyphoid  $\beta$  organism is culturally unlike the paracolon and resembles the typhoid bacillus, but produces a primary acidity and terminal alkalinity in milk. It does not coagulate milk, nor produce indol in broth. It ferments glucose, but not



lactose. The typhoid organism does not ferment glucose, nor produce terminal alkalinity in milk. Both the  $\alpha$  and  $\beta$  organisms, especially the latter, produce typhoid-like symptoms, and thus differ from paracolon and colon infections.

Over 100 cases are on record, a few under 12 years of age. The disease is probably by no means rare, even in the young. Possibly 5 per cent. of cases clinically typhoid are of this nature. Its geographical distribution is widespread. It occurs sporadically, in house epidemics and in general epidemic form. Sometimes there is a mixed infection with typhoid and paratyphoid organisms. The seasonal incidence, age and sex of the patients are those of typhoid.

*Symptoms.*—It begins with anorexia, headache, pain in the back and limbs, and irregular fever; sometimes vomiting and abdominal pain; rarely an initial chill. The tongue is more or less furred, labial herpes sometimes present, and there are the usual signs of malaise. Constipation is more common than diarrhoea. Abdominal distension is infrequent and due to unsuitable food. Rose spots come out in 50 per cent. The spleen is palpable in only half the cases though the splenic dulness is generally increased. Bronchitis is common; epistaxis, albuminuria and vomiting occasional. The urine may contain indican and give the diazo-reaction. Leucopenia is slight or absent. The pulse varies with the fever, usually 90-100 per min., and is sometimes slow and irregular, sometimes dicrotic. The fever resembles that of typhoid in its course, but is more intermittent and more apt to end by crisis. There is less anæmia and wasting than in typhoid, no delirium except in severe cases, and occasionally somnolence and apathy. The *course* is that of mild typhoid. Some cases are severe and a few are fatal. The duration is 3-5 weeks, perhaps only 10 days or as long as 9 weeks. Relapses are not uncommon.

The *complications* are hæmorrhage in about 5 per cent., rarely severe, pleurisy, broncho-pneumonia, endocarditis, phlebitis, meningitis, peritonitis, cholecystitis, pyelonephritis, cystitis, orchitis, suppurative arthritis, furunculosis and myositis.

The *diagnosis* is based on the agglutinating properties of the blood serum, the course of the disease, and the recovery of the specific bacillus from the blood or fæces. No case can be accepted as scientifically proved unless the organism is cultivated, the serum reactions obtained, and the absence of Eberth's bacillus certain. The serum does not agglutinate the typhoid organism. In 2 cases (Allen, Pratt) the  $\beta$  bacillus was recovered from the gall bladder; and in one of Pratt's cases from a suppurating testis after supposed typhoid. The general blood infection by the  $\beta$  organism is like that due to the typhoid bacillus in its symptoms, course and complications. So alike is it, that many physicians refuse to recognise paratyphoid fever as a specific disease, preferring to regard it as a mild variety of typhoid.



The anatomical changes in fatal cases are those of a general blood infection. Typical ulceration of Peyer's patches is lacking. In Van Ingen's case the spleen was not enlarged, and several ulcers were found just above the ileo-cæcal valve, with "deep punched-out appearance, extending in some cases to the peritoneal coat," but Peyer's glands seemed normal. In Wills and Scott's case extensive, superficial, ragged ulceration, without infiltration, was found a short distance above the ileo-cæcal valve. Typhoid fever can occur without changes in the agminated glands. Lazarus-Barlow states that not more than 20 cases of this type are on record and only 6 of them scientifically proved; possibly some were paratyphoid fever. In private practice the difficulty of diagnosis is great. Neither the parents nor doctor regard the child as sufficiently ill to warrant blood examination and cultures. The organism can be recovered from the stools. The *treatment* is essentially the same as that of typhoid but the diet may be somewhat more generous.



## CHAPTER LXVI.

### SPECIFIC FEVERS.

#### *Infection and Disinfection—Scarlet Fever.*

The infective disorders, so far described, are those in which definite causal organisms have been discovered. Of these diseases diphtheria and influenza are distinctly contagious, while syphilis, tuberculosis, typhoid and paratyphoid fever are communicable. Malaria, due to a protozoon, might also be included.

The specific fevers are those which run a definite course, uninfluenced by treatment, after a recognised incubation period. So far bacteriologists have been unable to prove that any one of the various organisms isolated by them is the true causative agent. The factors necessary for infection are a susceptible subject, and an infective agent in a sufficiently large dose. The external conditions must be suitable for its life and growth, and there must be a vehicle for its conveyance.

Susceptibility varies in different races, families and individuals, in the same individual at different times, in different diseases, and according to the mode of infection. Children are more liable than adults, the latter being frequently protected by a previous attack. Measles is the most infectious fever. The virulence of the virus varies in different epidemics and in the course of the same epidemic, becoming milder toward the end. It is modified by season and locality.

The virus is conveyed directly by the air currents or by direct contact, as in kissing or from infected hands, etc. Or it may be conveyed more indirectly by (1) doctors, nurses, attendants and animals; (2) infected milk, water or food; (3) by fomites, e.g. in various articles handled by the sick or contaminated in the sick-room, viz. toys, books, articles of clothing, wall papers, etc. During epidemics there are individuals whose susceptibility is so slight that the resulting illness may be trivial and entirely disregarded. These ambulatory, mild and unsuspected cases are often the source of outbreaks of diphtheria, influenza, typhoid fever and any of the true specific fevers. A further cause is the persistence of infectivity in the apparently cured. The diphtheria bacillus is found in healthy throats and often for a long time after an attack. Epidemics of typhoid have been traced to typhoid "carriers," patients whose urine or fæces still contain the organism.



*Prevention of Spread of Infection.*—The suppression of contact is the primary essential. Expired air, except on coughing and spluttering, is sterile. Hence the dangerous aerial zone round a patient is very small. The child is isolated at home or in a hospital.

For home isolation choose a medium sized room, without carpets or curtains and containing a minimum amount of furniture, at the top of the house and facing west, south-west or south. The bed must be narrow and of iron. A flat of 2-3 rooms, with lavatory attached, is the most suitable accommodation, for the nurse must be isolated as well. An open fireplace and open window afford the best ventilation. Keep down dust by dusting with a cloth damped with izal or other disinfectant, and clean the floor in a similar manner. A paraffined parquet floor and painted walls, washed twice a week, are ideal conditions.

Home isolation is superior to hospital treatment, if it can be carried out efficiently. Fewer secondary cases occur. Frequently it is defective or ineffective. There is a slight risk of the infection being conveyed by members of the household. Susceptible ones should be removed.

In hospitals all cases, whether mild or severe, are aggregated in wards. Occasionally there is segregation in single rooms or cubicles, but it is expensive in structural and nursing arrangements, and more trying for the patient. There is always the risk of a mild case becoming severe, from further infection; that, if an erroneous diagnosis has been made, the disease may be acquired in the ward; that secondary infection of the naso-pharynx may take place and increase the duration of infectivity; and that relapses or second attacks are more apt to occur from re-infection. Hospital isolation has not proved successful in limiting the prevalence of scarlet fever.

In order to prevent return cases do not encourage the child to mix or sleep with others on going home. Guard against chills, for catarrh of the mucous membranes is conducive to the growth of stray infective organisms. Disinfect the mucosa and provide separate towels and utensils.

The physician should visit his infectious cases last on his rounds, put on a long white overall before entering the room, avoid personal contact as much as practicable, and take special care against being infected by discharges when examining the throat, nose, skin, etc. On leaving the room he must disinfect his instruments and hands, washing with soap and a nail-brush, and using a lotion of lysol, or 1 in 1000 sublimate. The nurse must take every precaution against conveying the virus. She should use a nasal spray and an antiseptic gargle when nursing cases of diphtheria and scarlatina anginosa.

*Disinfection.*—The room is disinfected by free ventilation and washing with izal or cyllin 2 per cent. Sublimate lotion is poisonous and spoils metallic substances, permanganate stains, and phenol has an unpleasant



smell. Food utensils are put in a pan of water and boiled for an hour. Linen, towels etc. are placed for 24 hours in a wooden tub of water containing in each gallon 1 dr. of hyd. perchlor. 1, common HCL 2 parts, and then sent to the wash. Longer soaking and stronger solutions destroy the fabric. Or soak them in a solution of zn. sulphate. oz. 4, sod. chlorid. oz. 2, water 1 gallon, and boil for 2 hours. Saturated steam at 115° C. is the most satisfactory means of disinfecting articles not spoilt by moisture. Disinfectants have been standardised in relationship to carbolic acid. Thus for  $\beta$  typhosus perchloride of mercury has a co-efficient 20, i.e. is 20 times as strong, while the co-efficients of other disinfectants are cyllin 15, creolin 2·5, lysol 2·5, formalin 0·3, lysoform 0·1, sanitas 0·02. The price is a great consideration. Carbolic acid is 1s., cyllin 4s. per gallon. For the disinfection of excreta see typhoid fever (p. 918).

The room is finally disinfected by sheets soaked in formalin, 500 c.c. per 1000 cub. feet air space, to evolve gas; fumigation or spraying with formic aldehyde 2 per cent. It can be fumigated with powdered sulphur, put in a pan with red hot coal and stood on bricks over water, the room being moistened by steam and closed for 24 hours. Autan, a mixture of barium chloride and paraformaldehyde, is useful and merely requires the addition of water for evolution of steam and gas. The formalin-permanganate method, dependent on mixing pot. permang. 1 and formalin 2 parts, is dangerous for explosion may result. After disinfection the room should be papered and painted.

*The Coincidence and Interaction of Diseases.*—Two acute diseases may run their course without influencing each other to a recognisable extent. The total effect is generally one of increased malaise. Measles and diphtheria in the very young are almost invariably fatal. Measles may be coincident with scarlet fever or varicella; scarlet fever with varicella, variola, pertussis, diphtheria, typhoid or erysipelas; typhoid with paratyphoid or diphtheria, etc. In one child diphtheritic palsy and poliomyelitis were associated; in another, suffering with tuberculous meningitis, the meningococcus was isolated on lumbar puncture. A more common coincidence is an acute illness in the course of a chronic one. The beneficial influence of erysipelas on new growths, lupus, chronic eczema and chronic ulcers, has often attracted attention. Vaccination with calf lymph has been noted to modify pertussis and render it comparatively harmless, and sometimes has a good influence on eczema and chronic skin affections. Possibly in many cases the effect is merely one of coincidence.

## SCARLET FEVER.

Scarlatina is an affection of the skin and throat, with many complications. The disease is mild, apart from streptococcal infection. Complications and fatal cases are due to such infection and septicæmia.



It is doubtful whether there is a special family liability to infection, severity or immunity. Negroes are less susceptible than Caucasians. Epidemics may follow the re-assembling of schools after holidays. Both sexes are equally liable. The disease is infrequent in infancy. Susceptibility increases up to the sixth year and then slowly diminishes. About 1 per cent. of the cases are under 1 year of age, 50 per cent. under 6, and 90 per cent. under 10 years. It has been reported in the third week of life. Malnutrition, uncleanness and bad teeth predispose to a severe attack of the anginal type.

**Bacteriology.**—Edington (1886) isolated a diplococcus, streptococcus rubigenosus, and a bacillus. Class of Chicago (1899) cultivated a large Gram-negative diplococcus or tetracoccus from 300 consecutive cases, and from surgical scarlet fever. It was found by Schamberg and Gildersleeve (1904) in only 15 out of 100 cases. Curtois (1899) found an organism, allied to the *S. pyogenes* and *S. erysipelatosus*, in the urine of 42 out of 97 cases and in the heart blood after death. Klein (1887) has also isolated from the blood a micrococcus or streptococcus very like Edington's organism. An outbreak of scarlet fever at Hendon in 1885 was traced to cows with a specific, contagious and infectious disease apparently due to a similar organism. There is no positive proof that cows get scarlet fever. An epidemic of 400 cases in London and Surrey (1909) was traced to cow's milk. Human infection of the milk could not be proved and apparently the infective agent was derived from a newly calved cow. This was suggested as a cause by Sir W. Power (1882). Palmirsky and Zebrowski regard the *S. conglomeratus* as the specific organism, but up to the present there is no reliable proof in favour of any one germ. Many of the organisms found are due to secondary infection. In a mild epidemic Hektoen found streptococci in the blood in 12 out of 100 non-fatal cases. In a severe epidemic Jochmann found them in 25 out of 161 cases, and all died. Baginsky and Sommerfeld noted a general streptococcal infection in 82 fatal cases, and indeed it may be regarded as the constant cause of a fatal issue, though sometimes absent in fulminating cases. This organism (*S. pyogenes*) is constantly present in the throat. It is absent from the blood in a large number of cases.

**Mode of Infection.**—The virus is not transferable to animals. It can be transmitted a short distance through the air by coughing and sneezing. Contact infection, direct or indirect, is the chief mode of transmission. The virus is destroyed by the usual methods of disinfection. In the absence of sunlight and air it can remain alive outside the body for months, and can probably multiply if moisture is present. The contagium is present in the pharyngeal and faucial mucus; in the discharges from the throat and nose, and from the ears if infected; and possibly in the desquamation, urine and fæces. Mild cases and contacts distribute infection as readily as severe ones. Healthy persons have been known to act as carriers. Epidemics



have been traced to milk from infected farms and even to milk kept in a kitchen to which scarlet fever patients had access.

The throat contains the contagium in the early stages. The disease is infective in the pre-eruptive stage, as soon as the throat is affected. J. W. Stickler (1899) mixed some mucus with weak carbolic acid and injected it sub cutem into 10 children, setting up scarlet fever in all. The contagium may remain latent in the mucosa of the throat, nose and nasopharynx for a long time. Rhinitis is often present and intractable, though not necessarily infectious. Otorrhœa may be the supposed source of infection but, seeing that it is so often associated with rhinitis, the infective agent may be in the nose. Infection has also been ascribed to eczematous conditions of the nose, mouth, ears and anus, and to various complications such as empyema and suppurating glands.

Desquamation has been regarded as the main source of infection, but during recent years considerable doubt has been thrown upon it. The rash is an erythema, probably due to toxæmia, and the evidence that the squamæ contain infective germs is unreliable. There is no distinct evidence that the virus is present in the skin. Infectivity begins before desquamation and may continue long after it is completed. It is proved by the so-called "return cases" that a child who has been sent home from a fever hospital, after complete desquamation, may spread the disease. Many of these have been traced to uncured complications of the throat, nose and ear. Turner (Metropolitan Asylums Board Report, 1906) in an analysis of 1000 successive cases, found that return cases occurred in 3·27 per cent. of 397 without complications; 2·62 per cent. of 347 cases with rhinorrhœa; 1·54 per cent. of 256 cases with other complications. Of those with nasal discharge 180 were kept over 10 weeks in hospital. The general conclusion is that return cases are not due to coincidence; that they are often due to rhinorrhœa; and that late desquamation is little or not at all infective.

Apparently genuine cases have mixed with other children while still desquamating, even in the fourth week, without conveying the disease. Lauder of Southampton (1904) reported that 204 cases discharged while peeling gave rise to only 2 return cases. At Leicester 120 desquamating cases were discharged from hospital without any secondary cases developing. On the other hand infection has been traced apparently to the squamæ conveyed by letters, books, etc., but they may have possibly been contaminated by mucus from the mouth. According to Fowler of Pottsville, Texas (1903), casts from cases occurring in the spring of 1900 were preserved by the mother in a cardboard box and exhibited in February, 1901, to 2 parents and a boy aged 3 years. The child developed malignant scarlet fever on the fourth day and spread the infection to 6 other persons, of whom 2 were adults, and all died. There was no scarlet fever in the district. Here, too, the possibility of contamination with pharyngeal mucus cannot be excluded. At present it is advisable to regard the primary desqua-



mation as infectious and the later, or secondary desquamation, of the hands and feet as harmless.

The contagium is deposited on the mucous membrane, probably of the throat, and there develops and makes its way into the blood. In surgical or inoculation scarlet fever, the poison is deposited on the raw surface and passes directly into the blood. Some children are susceptible to this mode of infection, though apparently immune to infection through the mucous membranes.

*Duration of Infectivity.*—Infectivity exists from the onset to the end of the primary desquamation. It is slight within the first 24 hours, most active during the period of sore-throat and the height of the fever, and is prolonged by catarrhal conditions or complications. It is not improbable that nasal or faucial catarrh, due to the disinfecting hot bath and transference from the hospital, leads to the development of latent infection hidden in the folds of mucous membrane; just as in diphtheria the specific organism can sometimes be recovered for many months from the throat. As a rule infectivity does not last for more than 6 weeks. But absolute safety can never be determined. Nasal discharges must be viewed with suspicion. The infectivity of mucoid and purulent discharges may last for months. The contagium may remain in clothing, books, furniture, etc., for long periods of time, and can be shut up in wall papers by pasting a new one over the old. It appears to require moisture for its development.

*Quarantine.*—The child should not be allowed to mix with others for 6 weeks from the onset, 2 weeks after terminal disinfection, or longer if there is a persisting otorrhœa, nasal catarrh, or pharyngeal trouble. Sometimes the period is shortened to 4 weeks. In view of the uncertainty of the infectivity of squamæ, it is not advisable to permit a child to return to school until desquamation is complete. Previously he should have several soap and water baths, followed by sponging with carbolic acid 1 in 50 or bichloride of mercury 1 in 5000. The hair should be cut short and disinfected. R. Milne (1908) maintains that the spread of the disease can be entirely prevented by complete inunction with carbolic oil, 10 per cent., or eucalyptus oil, twice a day for 4 days, and once daily until the tenth day. The throat is swabbed with the carbolic oil every 2 hours for the first day. It would be rash at present to rely on such treatment.

Children exposed to infection should be quarantined for a week, unless they have only been in contact for a few hours with a very early case. Those living in the same house as a patient should not be allowed to mix with others, for they may act as carriers even without having a mild and unrecognised attack. They should use antiseptic gargles of Condy's fluid or lysol, 1 in 1000. A case in a hospital ward must be at once sent out, and the ward closed if the infection spreads.

*The Incubation Period* varies from 1-9 days. It is rarely more than a week, commonly 2-4 days, and occasionally only a few hours. If of longer



duration, it is probable that the source of infection has been overlooked or that the germ has remained latent in some fold of mucous membrane. It depends partly on the point of entrance, the virulence of the poison, and the susceptibility of the patient. Generally speaking a short incubation period indicates a severe attack. This does not apply to inoculation or surgical cases, in which the germ enters the blood stream directly and the incubation period may be only 6 hours.

**Symptoms.**—The onset is almost invariably abrupt and in severity is proportionate to the severity of the attack. In the adult the sequence of invasion is sore-throat, headache, fever and vomiting; in the child vomiting or convulsions, fever, and sore-throat. Vomiting is the first and most characteristic sign. It occurs once or twice; and may be very frequent and severe, even in mild cases. It is associated with sore-throat, shivering, headache, rapid rise of temperature, and general malaise in proportion to the severity of the attack. Some cases are so mild that there are no particular symptoms. Others are so severe that the patient dies before the characteristic rash or sore-throat develops. Mild cases with insidious onset, slight malaise and little fever, may be followed by severe nephritis. The face is flushed and perhaps swollen. The sore-throat precedes the rash. The rash may be the only sign, or it may be so faint or transitory as to escape notice. A delayed rash is often associated with marked angina. The child may only vomit once or twice, or the vomiting may be severe and persistent for even 24 hours. Occasionally epistaxis, diarrhoea and pains in the limbs are present at the onset.

With rare exceptions the *temperature* remains up until the rash is out. It reaches its maximum on the first to the fifth day, generally the second or third day, remains up for a variable period, and subsides by lysis, or perhaps by crisis in 2 or 3 days. It usually persists for about a week. Occasionally it does not rise until the rash has come out. Subsequent fever indicates complications.

The *rash* generally appears on the second day, almost invariably 12-36 hours from the onset, and is rarely delayed beyond the third day. The symptoms increase as the rash develops and usually subside as it fades. It is a punctate erythema; minute, brightish or dusky red spots on a general erythema, as if the skin had been sprinkled with cayenne pepper, giving it a lobster-coloured appearance. The erythema may appear first, but more usually is simultaneous with the punctæ; and the relative proportions vary in different cases. The rash comes out first on the neck at its junction with the sternum and spreads peripherally in all directions, especially on the inner aspect of the limbs. It sometimes begins behind the ears or as erythema of the forehead and cheeks, avoiding the regions round the mouth and thus producing the "circumoral pallor." It is most marked on the parts protected from exposure, notably the groins and axillæ. It spreads very rapidly, and is usually fully developed and reaches the hands



and feet in 24 hours or less ; but it may be delayed or driven in again by exposure to cold, and brought out by heat or a hot bath. A case, diagnosed as diphtheria in a hospital out-patient department on account of the condition of the throat, may be typically scarlet fever as a result of the warmth of the ward. The rash may become distinctly papular ; chiefly on the fore-arms and legs. If so, these papules persist for some time after the erythema fades, are of diagnostic value, and disappear gradually. The skin is hot and burning, and perhaps itches. A certain amount of swelling, chiefly of the face and hands, is seen in bad cases. On pressure the rash leaves a yellowish discoloration ; later on, a palish brown. It remains at its maximum until the third day of the illness ; and lasts for 3-7 days, occasionally 11 days, but very rarely a longer or shorter time. It disappears in the same order as that of its development.

Atypical rashes may be limited to the groins and inner aspect of the arms and thighs. The rash is rarely completely absent, a little is probably present in the axillæ and groins. Sometimes it closely resembles the rash of rōtheln, occasionally it is like true measles, and in malignant cases it may be dusky, vesicular or hæmorrhagic. Milk-white patches, due to vaso-constriction, may be noted.

*Desquamation* is branny. It commences as minute points and causes a pin-hole or worm-eaten appearance of the skin. Then it becomes flaky, in the form of fine scales on the neck and chest, and occasionally between the fingers. It begins on the fourth to sixteenth day, generally the seventh to tenth, as soon as the rash has faded. It bears no absolute relative proportion to the rash, but as a rule is most marked if a characteristic vivid eruption is noted. It is most extensive on the palms and soles where the rash is least visible and the epidermis thickest. It does not occur in every case. Desquamation is completed on the trunk in 3-5 weeks from the onset, but on the hands and feet may take 8-18 weeks. Large portions, like the fingers of a glove, may be cast off. The new epidermis is thin and gives the skin a pinkish colour. This is most marked at the tips of the fingers, which peel first, and affords a characteristic contrast with the thick grey patches in course of separation. The peeling alone may afford a reliable means of diagnosis in the absence of a history of an attack.

**Varieties.**—The disease varies greatly in severity. For convenience of description cases may be divided into groups, although there is no strict line of demarcation between them.

1. *Scarlatina Simplex* is of mild, medium or great severity. In the mild cases there is no complaint of sore-throat, a slight and indefinite rash, and a temperature little or not at all raised. Thus two children in succession had a slight rash somewhat like that of mild rōtheln, T. 99° F. for a few hours, and no vomiting or constitutional symptoms. Another child developed severe *S. anginosa* and a nurse typical scarlet fever. Some mild cases are characterised by slight fever, rash and a little redness of



the throat, with or without subsequent desquamation; in others the rash is absent; and in a few desquamation is alone noted. In cases of medium severity the attack is ushered in by vomiting, T. 101-103° F. moderate sore-throat and typical rash. The fever lasts for about a week. In the severe type the onset is more acute, vomiting may be frequent, possibly convulsions occur, and the temperature rises to 103-105° F. The rash is intense and comes out early, delirium and restlessness are present, and the fever is more prolonged.

2. *Scarlatina Anginosa vel Septica* is a variety in which the throat symptoms are predominant and severe. It is most common in hospital cases, the ill-nourished and the uncleanly. It often begins mildly, but on the third to the fifth day the throat signs become exaggerated, the temperature rises higher, and the cervical glands enlarge. The rash becomes petechial or hæmorrhagic, especially on the knees and elbows, and is, therefore, of a septic type. Diarrhœa is not infrequent.

The *throat* in mild cases shows nothing but a little redness and congestion of the fauces; possibly a punctate or erythematous rash on the soft and hard palate; or a ring of bright redness of the edges of the palate and pillars of the fauces. In more severe ones there is a vivid, carmine-like redness, intense and dusky, of the whole of the fauces, palate and buccal mucosa, with considerable swelling and pain on swallowing. The tonsils swell, perhaps meet in the middle line, and are more or less covered with dirty yellow secretion, somewhat like that of follicular tonsillitis at first. The pharynx becomes full of muco-pus, the glands enlarge, the nose discharges, and the tonsils ulcerate in a few hours. Thus the 3 stages are those of swelling, exudation, and ulceration. In *S. anginosa* there is great swelling, and ulceration of the tonsils and palate is rapid and extensive, sometimes gangrenous. The tonsils, uvula and fauces become covered with a soft, white, pultaceous, easily detachable deposit, sometimes pseudo-membranous. The whole buccal cavity may be involved and the mischief may extend to the pharynx, nose and ears, which become filled with foul secretion. The ulceration may be deep and cause severe hæmorrhage. Occasionally the throat at the onset shows extensive swelling like that of erysipelatous inflammation, without any sloughing or ulceration. Sometimes it presents all the appearances of diphtheria within 12 hours.

Secondary to the throat symptoms is the rapid development of *cervical adenitis* and infiltration of the cellular tissue, forming the anginous collar or "bull-neck." The breathing is interfered with by the pressure and the head is thrown back. These glands frequently suppurate and occasionally there is extensive sloughing.

The general symptoms of the anginal form are those of septicæmia of faucial origin. The breath is offensive; the mouth intensely congested, perhaps ulcerated and the discharge offensive; and the nose is similarly affected. Food is taken with difficulty because of anorexia, local con-



ditions and apathy. The temperature is high and prolonged, delirium common, and a typhoidal condition may supervene. Death takes place from septicæmia, pyæmia, or some septic complication, in 7-14 days or less, or perhaps not for weeks. Streptococci are found in abundance in the fauces, glands, blood and tissues.

3. *Scarlatina Maligna* or *Toxic Scarlet Fever* is the most fatal of all varieties. It is due to a large dose of a virulent poison, or extreme susceptibility. Its frequency varies in different countries and different epidemics. The symptoms are those of toxæmia, mainly cerebral. It is generally ushered in acutely with convulsions, vomiting, and the rapid development of cyanosis, stupor or coma, muttering delirium, tremor, prostration and hyperpyrexia. Prostration and vomiting may be marked and persistent from the onset. The pulse is feeble and frequent. The temperature may reach 106-108° F. in a few hours and the patient die comatose within 24 hours, before the rash appears. The frequency of respiration and cyanosis suggest pneumonia. Pyrexial changes only are found post mortem. In a less severe type life is prolonged for a few days. The throat is red, œdematous and covered with dry sticky mucus; the submaxillary glands are a little enlarged; the rash vivid and sometimes blotchy. Occasionally cases pass into the septic type and develop green foetid diarrhœa. Quite three-fourths of these malignant cases die.

4. *Scarlatina Hæmorrhagica* is a sub-variety of the last type. The epidemic malignant purpura of the older writers was scarlatina. Hæmorrhages take place into the skin and kidneys, and from the mucous membranes. Similar hæmorrhages may occur in cases of medium severity, and be indicative of secondary toxæmia.

5. *Surgical or Inoculation Scarlatina*.—Apparently individuals during the puerperium and those with open wounds, abrasions, sores, burns, etc., are peculiarly susceptible to this disease. It may be acquired in the ordinary way by such a patient, or be due to direct inoculation through the raw surface. Care must be taken to exclude the vasomotor and toxic erythematous rashes, due to serum disease, intestinal toxæmia or soap and water enemata; and the septic rashes liable to follow burns, vaccination, surgical operations and other injuries. If the scarlatina is merely coincident and of the acquired type, it differs in no way from the ordinary attacks. Only inoculation scarlatina should be regarded as genuinely surgical. It has followed the simplest operations, the scratching of pimples and vaccination. According to Henoch the incubation is 4-7 days in operation cases. It may be only a few hours. A membranous exudate appears on the edges of the wound, with secondary swelling and redness of the surrounding skin and adenitis. The rash spreads to the rest of the body, but it does not always begin locally. The wound improves after the rash reaches its maximum, and desquamation begins round it. The throat symptoms are slight. There is no sore-throat in the cases of septic rash.



**General Symptoms.**—The patient is flushed, noticeably so on the cheeks in comparison with the circumoral pallor. The skin is burning and dry. The lips are dry and in bad cases covered with sordes. The mouth is partly open if there is much angina. The breath is said to have a peculiar, sweet, almost aromatic, acetone-like odour in the early stages. In the presence of angina and ulceration it is offensive, foetid, and even gangrenous.

The *tongue* is characteristic at certain stages. The early “strawberry tongue,” is swollen and covered with thick, moist, creamy white fur through which the fungiform papillæ stand out prominently. It looks somewhat like an unripe strawberry. It begins to peel on the second or third day, perhaps not till the fourth, and in another 2 days it has a bright, raw, red, clean appearance with red prominent papillæ like that of a ripe strawberry or raspberry. The peeling begins at the tip and edges, spreads backward until the tongue is entirely denuded of epithelium, and is generally complete on the fourth day. The tongue becomes normal in about 7-10 days.

The *pulse rate* is a valuable diagnostic point. It is often remarkably frequent, unduly so in proportion to the temperature, and even 120-180 or more per minute in comparatively slight fever. Infrequency and irregularity are common in convalescence. The breathing is increased in proportion to the fever. The temperature is high in proportion to the rash and hyperpyrexia may occur. Recovery has followed a temperature of 107.6° F. The condition of the glands depends on the throat.

The *blood* shows leucocytosis of polymorphs, and 20 per cent. reduction in hæmoglobin and the number of red cells (Mackie, 1901). The leucocytosis is present in the mildest cases; is most marked after full eruption, and persists for a few days after the fall of temperature; and does not depend on the height of the fever. It is increased in severe rash, bad angina, and sepsis. Its absence in severe cases is a bad sign. From 5-15 per cent. of eosinophiles are present, probably due to the skin being involved.

**Complications.**—The post-scarlatinal infections rarely begin before the fourteenth day or later than the twenty-eighth day, but no patient is safe until the seventh week. They are ushered in by a rise of temperature which lasts for a variable period and drops by lysis. Such a rise of temperature may occur without signs or symptoms. The complications are due to general infection, or direct extension from the throat. The common throat complication is the streptococcal infection, already referred to, in which the exudation resembles follicular secretion or false membrane even within 12 hours of the onset. A diphtheritic looking throat during the first week of the disease is almost invariably streptococcal. It extends to the posterior nares and causes adenitis. It rarely spreads to the larynx and is not followed by paralysis. Later on, say after the disappearance of the rash, such a throat is probably true diphtheria, liable to involve the larynx and to produce the usual effects of diphtheria. It is not an uncommon complication, and patients seem very susceptible to it during



convalescence. Epidemics of post-scarlatinal diphtheria and of diphtheria in scarlet fever wards are not infrequent. Some of these cases yield both streptococci and diphtheria bacilli on culture. Ulceration, necrosis and gangrene are not uncommon. The tonsils become greyish black masses of necrotic tissue, sloughing and giving rise to foetid odour and discharge. The cellular tissues in the neck are involved, severe and fatal hæmorrhage may occur, or there is asthenia and profound cachexia. Gangrene of the tonsils and fauces may occur in the cachetic and is invariably fatal.

Perforation of the soft palate is a rare terminal stage of ulcerative angina and is due to cellular necrosis. It is usually bilateral, appears in a few hours in the severest cases, and is very resistant to treatment.

A slight initial adenitis is nearly always present. The epitrochlear glands are enlarged, even in the mildest cases, and the inguinal glands in 80 per cent. (Baum, 1907).

Secondary adenitis is due to infection from the mouth and throat. It may be extreme and set up a diffuse, brawny, œdematous cellulitis of the neck. When a single gland only is affected, it is much more likely to suppurate and break down. The pus may make its way into the mediastinum and cause purulent pericarditis, empyema and pyæmia. Post-scarlatinal adenitis generally affects the submaxillary lymph nodes, and is usually limited to one gland on one side only. It is ushered in with high fever for a short time, 24 hours or less, and the swelling subsides in 2-6 days. It is rarely as marked as in the primary stages of the disease and is less likely to suppurate. It is often associated with albuminuria.

Rhinitis occurs in 5-10 per cent., is easily recognised by the discharge, and may persist for months. Necrosis of the nasal cartilage is rare. Otitis media (p. 994) occurs in 15-20 per cent.

Albuminuria is present in 15 per cent. of all cases, but in less than 5 is there an acute nephritis. It is due to the cloudy swelling from the fever, or a mild and transient catarrhal nephritis in the first week. A small amount of albumin and a few hyaline casts, and occasionally renal epithelium, granular casts and a few red and white cells are found in the urine. True scarlatinal nephritis is glomerular in type (p. 576).

Hæmorrhagic purpura is sometimes associated with acute nephritis or grave fatty degeneration, but albuminuria is not always present. It may occur during convalescence, and is probably toxic in origin, a sequel of severe angina and pyrexia. The bleeding takes place subcutaneously and from the mucous membrane. It is very fatal, even within 24 hours.

The cardiac muscle may be soft and friable as early as the fourth day, and sometimes there is acute diffuse myocarditis. Dilatation is due to cloudy swelling, anæmia, myocarditis, and the increased blood pressure in nephritis. It may be acute and cause sudden death. Endocarditis and pericarditis are rare. They are due to septic conditions or scarlatinal rheumatism. Endocarditis may occur in mild cases and produce permanent



damage. The functional murmur of dilatation must not be ascribed to endocarditis. Embolism, and its effects, and malignant endocarditis are not unknown.

The pulse usually drops to or below normal when the temperature comes down. It may fall quite suddenly and before the fever, or may remain high though the fever has subsided. A later acceleration, with irregularity, apical murmur and accentuated pulmonary second sound, suggests a cardiac affection.

Gangrene of the extremities is rare, and due to thrombosis or endarteritis. It may come on as early as the sixth day. It is "dry," and usually, but not always, symmetrical and bilateral. If limited to a limb, it may terminate favourably after amputation. Both legs were amputated above the knees in a boy, aged 4 years (Pearson and Littlewood, 1897). The buttocks and thighs are sometimes affected simultaneously or successively and the process ends fatally.

Laryngeal affections are rare except in diphtheritic complications. Broncho-pneumonia, pneumonia and empyema may occur in the septic type, and serous pleurisy and œdema of the lung in nephritis.

Obstructive jaundice is due to intercurrent disease or, exceptionally, is septic in origin and fatal. Diffuse inflammation may extend from the pharynx to the œsophagus and cause ulceration and stenosis. Vomiting after the onset is frequently uræmic. Diarrhœa occurs in uræmia and sepsis. Peritonitis is very rare and is part of a general streptococcal infection late in the disease. It is purulent and diffuse, but not always fatal.

Convulsions may occur at the onset. In the course of the disease they are generally uræmic or meningeal in origin, e.g. meningitis, hæmorrhage, thrombosis or embolism. Aphasia, hemiplegia, encephalitis, peripheral neuritis, and epilepsy are exceptional.

The joint affections are rheumatic or pyæmic. Scarlatinal rheumatism, or synovitis, is uncommon under 5 years of age. It comes on at the end of the first or beginning of the second week; affects the upper rather than the lower extremities, the wrists and hands chiefly; and may involve the elbows and knees, and even all the joints. There is moderate swelling, pain and redness, and slight fever. It runs a mild course of a few days' duration, and rarely suppurates. It differs from rheumatism in the absence of acid sweats, in not responding to salicylates, and in not jumping about from joint to joint. It varies in frequency in different epidemics, has little or no tendency to relapse, infrequent heart complications and no special predilection for rheumatic patients. Chorea is extremely rare. Probably it is due to toxæmia, though true rheumatism may occur during convalescence. Pyæmic arthritis is often multiple and associated with pyæmia. It may start as an epiphysitis. The large joints are affected and suppurate.



Four or five weeks after the onset a transverse linear groove or slight ridge may appear at the root of the nails, being most marked on the thumb. It travels up the nail as it grows, reaching the free edge in about 6 months. It is more marked in adults than in children, after a severe than a slight rash, is due to nutritional disturbance, and may occur in other febrile states. In rare instances the nails are shed. Sometimes the skin shows atrophic striæ, similar to the "striæ patellares" occasionally seen after typhoid fever.

**Diagnosis.**—Typical cases are easily recognised, but those which are very mild, extremely malignant, or atypical in character and course may be impossible of diagnosis. The occurrence of desquamation, or of secondary cases, may be the only positive indication, and even these are not absolutely reliable. The desquamation may be uncharacteristic, may be absent, and can occur in other affections. Secondary cases may be due to other modes of infection. Considerable importance should be attached to undue frequency of the pulse in mild and apyrexial cases, provided it is not due to nervousness; and to the condition of the tongue in relation to the rash and the duration of the illness. Frequent vomiting during the first 24 hours may be the only sign that a sore-throat is scarlatinal. If the patient dies before the rash or sore-throat develops, the diagnosis is impossible. The nervous and toxæmic symptoms may suggest pneumococcal infection, cerebrospinal meningitis or some severe cerebral disease.

Ordinarily the diagnosis is based on the acute onset with vomiting, the rapid rise of temperature, the appearance of the throat, the unduly frequent pulse, the characters of the rash on the second day, and later on, the tongue, course, complications and peeling. The "throat" may be mistaken for simple catarrh, acute tonsillitis, follicular tonsillitis or diphtheria. Scarlatiniform rashes may occur in these affections, but the date of appearance and the distribution are often distinctive. A diagnosis must never be based on the rash alone. There is especial difficulty under the age of one year for the skin is so easily reddened. The erythema must not be mistaken for that due to fever, liniments, poultices, scalds, burns and sunburn. The erythema of influenza may be associated with faucial catarrh, enlarged and painful cervical glands, fever and aching pains. It comes out on the second or third day, and may be followed by branny desquamation, though it does not appear to be ushered in by vomiting. The cases closely resemble mild scarlatina. Erythematous rashes may occur in the prodromal stages of measles, variola and varicella; in diphtheria, serum disease and typhoid fever; after soap and water enemata; from drugs such as belladonna, antipyrin and quinine; and in urticaria, and various forms of sepsis and toxæmia. In mild cases it is diagnosed with extreme difficulty from the rash of rōtheln, aberrant types of measles and erythema infectiosum. Rhinitis, otitis and adenitis are strong evidence of a recent attack of scarlatina. Peeling, albuminuria and changes in the nails may be present.



*Fourth Disease.*—Filatow (1885) described under the name of “rubeola scarlatinosa,” and Dukes (1900) under the name of “fourth disease,” an affection intermediate in character between scarlet fever and rōtheln. Like the latter disease its incubation period is 9-21 days, and there are often no symptoms though the rash is copious. It may be ushered in with slight sore-throat, and occasionally malaise for some hours, headache, anorexia, drowsiness, chilliness and backache, but no vomiting. According to Dukes the rash is bright rosy-red, somewhat raised, and diffused over the whole body in a few hours. The amount of desquamation is variable, and has no relation to the severity of the rash, and it takes place in the form of small scales rather than flakes, for a week or two. The temperature is not raised in proportion to the rash; the pulse rate is not unduly increased; and the characters of the tongue, conjunctivæ, throat and glands, and the course of the disease are almost identical with rōtheln. Klein (1904) states that the incubation period is a few days to 14; and the onset is sudden, with slight signs of cold in nose and eyes, deep faucial redness, and submaxillary adenitis. The tongue on the third day is like that of scarlet fever. The rash appears on the face first, then on the back and abdomen, seldom on the extremities, lasts for 1-3 days, and is not followed by desquamation. These descriptions are not identical. Dukes maintains that he has seen scarlet fever both before and after this disease. The evidence is in favour of some epidemics being cases of mild scarlet fever. The absence of definite faucial inflammation, of peeling of the tongue, and of kidney disease, and the subsidence of the fever on the third or fourth day, are quite frequent in scarlatina. The apparent lack of infectivity after the third week is not of much assistance, seeing that it has been noted in scarlatinal patients even while desquamating.

**Course and Prognosis.**—Both depend on the variety and severity of the disease, and the prevailing type of epidemic. It may be extremely mild or peculiarly malignant. After a series of epidemic years, its virulence declines until it becomes a very mild affection. The mortality varies from 4-40 per cent. It is much higher under 2 years of age than at any other period of life, and still very high up to the end of the fifth year. In recent years the mortality during the first 5 years of life has been 5-7 per cent., in the second 5 years 1-2, and in the third 5 years under 1 per cent. Fatal cases die from acute toxæmia in the malignant or toxic type; from pyæmia or septicæmia in the severe anginal and later stages of the toxic variety; or from some complication of a severe attack, e.g. pneumonia, nephritis, or throat mischief; or from nephritis in late stages, sometimes even after a mild attack.

The prognosis must be guarded as to severity and the probability of sequels, which are impossible to prevent. Many depend on the state of the mouth. Wait until the third day before pronouncing definitely on the severity. Cases are usually regarded as milder than they really are. The



amount of the rash varies directly as the severity in many cases, but by no means in all. The temperature is not a reliable guide, though as a general rule the severity varies with the height of the fever. It may be moderate in a very malignant case, and up to 105° F. in comparatively mild attacks. A fall of several degrees does not always indicate improvement. The ordinary mild case has a little fever for a few days, perhaps only 1 day, while a more severe attack may have fever from 1-3 weeks, the temperature falling by lysis. An undue rise of temperature in the course of the disease may indicate complication. In a case of medium severity the symptoms last 4-7 days.

The character of the first sound of the heart is a measure of the severity of the toxæmia. Cold extremities and cyanosis show heart weakness. A very high temperature, high pulse rate, cardiac debility, gastro-intestinal and nervous symptoms, and great angina are bad signs. Hyperpyrexia may prove fatal.

Nephritis may occur in the mildest cases, but is most prevalent if there are streptococcal complications, i.e. many such cases are streptococcal nephritis. With increasing age the mortality from scarlatinal nephritis decreases from about 2 per cent. under 5 years of age to 0·2 per cent. over 15 years (Turner), and four-fifths of all cases get well. Possibly it may be a cause of chronic interstitial nephritis in later life.

**Relapses and Second Attacks.**—Relapses occurred in 1·09 per cent. of 14,143 cases in the Metropolitan Asylums Board Hospitals (Sloan, 1903). Ordinarily an attack confers immunity for the remainder of life but second attacks are not rare, and some patients are extremely susceptible. The liability to erroneous diagnosis, notably rōtheln, must not be forgotten before accepting a case as one of relapse or recurrence. The first attack should present a characteristic onset, angina, rash and strawberry tongue. So-called relapses may be due to temporary abeyance of the rash for a few days, perhaps on account of exposure to cold; to sepsis, especially in anginal cases; or to food poisoning, an enema or drug. Apart from these causes there are a certain number of cases in which a genuine relapse occurs, just as in typhoid fever. It is ushered in by vomiting, sore-throat and fever, as in the first attack. It is usually mild and of short duration; sometimes more severe, quite as liable to complications, and even fatal. Relapses and second attacks are more frequent in hospitals than in private practice. They may occur in mild cases after admission to hospital, generally coming on in 1-3 weeks. They must not be put down to contraction of the disease in hospital in a case of mistaken diagnosis, and are probably due to a larger dose of a more virulent poison, the patient being insufficiently protected. Such relapses have occurred at intervals of 14 days to 6 weeks after the first attack. Relapses in isolated cases have been ascribed to auto-infection, re-infection from profuse growth of the contagium in the



throat or naso-pharynx, but it is difficult to understand how such infection occurs, or why, if it does occur, relapses are not much more frequent.

**Treatment.**—The child is kept in bed until the temperature has been normal for a week, or for 3 weeks from the onset in even mild cases. The clothing should be light ; linen during fever and flannel when the temperature is down. The *diet* is limited to milk and milk foods for 3 weeks or until there is no fear of nephritis. Some physicians insist on a milk diet for 4-6 weeks, or until peeling is over, giving plenty of water and fruit juices to keep the urine alkaline. Others allow solid diet as soon as the fever has subsided, and the patient can swallow. It is doubtful whether meat and soups are injurious, even if there is nephritis.

The bowels must be kept open by calomel and saline cathartics ; an enema being given at once if the temperature is high. Warm baths reduce the temperature and quiet restlessness. For high fever, especially if there is hyperpyrexia, a typhoid state or cerebral symptoms, ice to the head, sponging and cold packs are necessary. Tepid sponging and bromides are good for sleeplessness ; phenacetin or an ice bag for severe headache.

No special treatment is required for the rash. If the itching is severe inunction with 5 per cent. ichthyol ointment is of value. It subdues the swelling of the skin, relieves the itching, reduces the nervous irritability, usually brings down the fever 3-4° F. within 2 hours, and improves the general condition. Tepid sponging, weak carbolic lotion, warm carbonate of soda lotion, gr. 10 to 1 oz. with a little mucilage, and rice powder are also useful. Eucalyptus oil is said to prevent the spread of infection, if rubbed in universally. As soon as the rash and fever have subsided, peeling should be encouraged by means of a warm bath daily with plenty of soap and water, followed by inunction with ichthyol ointment 1-5 percent., carbolised vaseline or carbolised oil 5 per cent., salicylic acid ointment 1 per cent., or borated vaseline 5 per cent. An ointment of resorcin 1, lanolin 6, and ol. sesami 2 parts, and a 5 per cent. resorcin-salicylic superfatted soap, are recommended by Jameson to hasten desquamation. Carbolic preparations must be used with caution as they are apt to irritate the skin.

The throat and nose are treated by ordinary methods (pp. 234, 373), especially formamint lozenges and swabbing. Attention to these parts may prevent otitis, but the treatment is difficult to carry out in many children. Cold compresses to the neck every 3 hours relieve throat affections. The mouth must be kept moist and free from mucus by frequent drinks of lemonade ; and borated vaseline applied to the lips and nose. If there is extensive ulceration, hot applications externally and ice to suck, or sips of hot water, afford relief. Tracheotomy may be needed for great oedema.

Toxic cases are treated by hydrotherapeutic measures, and septic cases by Moser's serum or Marmorek's polyvalent anti-streptococcal serum. Moser's serum is expensive, difficult to obtain and of doubtful value, though it is said to reduce the mortality in hospital cases from 15 to 9 per cent.



One dose only of 25-100 c.c. is given. In 8-12 hours the temperature falls rapidly and the pulse is less frequent, the rash does not develop or fades more quickly, the general malaise is less, and restlessness, delirium and somnolence usually disappear. Stimulants can be used freely in toxic and septic cases, and for cardiac weakness.

For otitis instil a few drops of warm 5 per cent. carbolised glycerin 2 or 3 times daily, and apply hot or cold compresses. Puncture the drum if there is much pain and high temperature. For otorrhœa instil hydrogen peroxide 3 volume strength.

Rheumatic complications are mild, transient, and require no special treatment. The prophylaxis of nephritis consists of confinement to milk and farinaceous diet, and bed for 3 weeks, and to the house for another 2 weeks. Give plenty of diluents and encourage excretion by the kidneys. Encourage desquamation. Spray the throat with chlorine water or Listerine. Give alkalies and laxatives internally. Urotropine is of no special value. Uræmia is treated by saline injections, high rectal irrigations and venesection.

Biniiodide of mercury gr.  $\frac{1}{24}$ - $\frac{1}{4}$  every 4 hours ; carbolic acid internally ; bisulphite of soda m. 3-10 of the saturated solution, two-hourly ; and 1 oz. doses hourly of decoction of cinnamon, have been claimed by different practitioners as specifics. There is, however, no special drug treatment, and reliance must be placed on the general methods adopted in fever. Expectant treatment gives the best results. Care is needed during convalescence, to prevent cardiac dilatation and nephritis. Undue exertion and chill must be avoided. Iron, arsenic and cod-liver oil are the best tonics. Persistent otorrhœa and rhinitis must be carefully treated as possible sources of infection.



## CHAPTER LXVII.

### SPECIFIC FEVERS.

#### *German Measles—Measles—Erythema Infectiosum.*

**German Measles**—*Syn.*: *Rötheln*—*Rubella*—*Rose Rash*—*Spring Rash*—*Epidemic Roseola*—*Hybrid or False Measles*.—*Rötheln* is a mild and feebly infective fever which occurs in epidemics, simulating true measles or scarlet fever. With these diseases it has often been confounded. It was described by de Berger in Germany in 1752; by Maton in England in 1815; and clearly differentiated by Wagner in 1834.

It occurs at all ages, but is rather more frequent in the later half of childhood than in the earlier half. Nurslings are rarely affected, but cases have occurred within the first 3 weeks of life and intra-uterine infection has been recorded. After the age of 10 years it is more frequent than measles. It is most frequent from March to June and at its maximum in May. Epidemics sometimes occur in the autumn.

The *incubation period* is usually 14-17 days. Most cases begin on the fifteenth or sixteenth day, a few as early as the twelfth or as late as the twenty-second. Contacts must therefore be quarantined for 23 days. The infection is usually direct. It may be conveyed by intermediaries, objects or persons. It is concentrated in closed rooms, schools, etc. The disease is mainly infectious in its early stages, especially at the height of the rash, feebly so and for a short time only.

**Symptoms.**—Its symptomatology is much the same as that of varicella, excepting as regards the rash. The prodromal period is often absent and rarely lasts for more than 24 hours. Sometimes there is a slight degree of fever, up to 101° F., malaise in proportion to the fever, headache, suffusion of the eyes, sore-throat, cough, and some catarrh. Occasionally the neck is stiff, and the cervical and occipital lymph nodes enlarged. The catarrhal symptoms are present in 30-50 per cent., the conjunctiva being injected, and the naso-pharynx involved. A few attacks are ushered in by shivering, giddiness, coryza, and pain in the back; and exceptionally by anorexia, nausea and vomiting, headache, drowsiness, convulsions, delirium and rigors, or epistaxis.

The temperature is normal in half the cases; or may rise to 100-102° F., rarely to 104-106° F. High fever is accompanied by restlessness, sleeplessness and delirium. The fever is highest as the rash reaches its maximum



and falls as the rash fades, being proportionate to its severity. Its duration varies from a few hours to 4 or 5 days. It may fall abruptly as soon as the rash appears, but generally subsides gradually with its disappearance. The degree of malaise depends on the fever.

The *rash* is out of proportion to the general symptoms. It comes out on the first day, within a few hours of the onset, and may be the first sign of illness. It appears first and chiefly on the face and scalp, at the junction of the scalp and forehead, on the region round the mouth and invading the circumoral region right up to the lips, on the cheeks or behind the ears. It spreads rapidly over the body, especially the chest and back, and attains full development within 24 hours. It may cause slight facial œdema, especially about the eyelids. It may appear on the palms and soles. It often disappears from the face, as it comes out on the trunk and there coalesces into a scarlatiniform rash. It may appear on the chest first, or simultaneously over the whole of the body with a special predilection for the cheeks, neck and under the chin, the buttocks and external aspect of the thighs. Usually the extremities are the last parts affected. It appears on the roof of the mouth which looks dry, mottled and dusky red.

The most common type is a *morbilliform* or maculo-papular rash. The spots are rather larger than the punctate spots of scarlet fever, and smaller than the papular ones of measles. They are often minute at first, increase in size, and may coalesce. They vary in size, are discrete, and pale pink or rose-red in colour, sometimes resembling spots of red ink on blotting paper. Coalescence produces raised blotchy patches on the face and a little swelling of the features. This rash often itches.

A much less common type is the *scarlatiniform* rash. There is a diffused redness which suggests scarlet fever, but some of the maculo-papules can be found on the extremities and perhaps on the forehead. It may itch. Urticarial patches may be present in both varieties.

Many intermediate types are seen. Occasionally a temporary or abortive rash is followed in 2 or 3 days by a typical one. The morbilliform rash is sometimes dark red, and rarely hæmorrhagic. It is less dusky than measles and does not coalesce into crescents. It lasts 1-4 or even 8 days, usually 2 or 3, and fades gradually in the order of eruption. It fades much more rapidly than that of measles; and frequently causes slight mealy or branny desquamation for a few days, and a little temporary pigmentation or discoloration of the skin. The desquamation may be profuse. Secondary rashes, probably relapses, occasionally occur.

The tongue is clean, or a little furred in proportion to the fever. It has been known to peel. The pulse rate depends on the temperature. Slight catarrhal symptoms of the throat, nose and conjunctiva are frequent, with itching, smarting, lachrymation and even photophobia. A secondary sore-throat may appear about the fifth day.



The glands behind the ears and posterior border of the sterno-mastoids are almost always enlarged, perhaps before the eruption. Occasionally the axillary, and more often the inguinal glands, are also affected. They are movable, tender, hard and shotty, like peas or equal to horse-beans in size, and do not suppurate.

**Complications** are practically unknown. Acute laryngitis is the most severe and fatal one. Death has occurred from pneumonia, and from mild arthritis with sudden heart failure. The catarrh may spread to the bronchial tubes and produce lobular pneumonia. Rare complications are albuminuria, diarrhoea, acute enteritis, otitis, joint pains and arthritis. Some of these may have been of scarlatinal origin. The disease is almost invariably harmless.

**Diagnosis.**—Many cases are wrongly described under this name. It is most likely to be confounded with mild or abortive measles. It is doubtful whether cases need ever be mistaken for scarlet fever, but the converse error in diagnosis is excusable. The difficulties may be insuperable. I have known three physicians pronounce a septic rash to be scarlet fever, measles, and German measles. From measles it is differentiated by its mild catarrhal symptoms, post-auricular and post-cervical adenitis, the mild invasion, early appearance of the rash, absence of buccal spots and of diazo-reaction, and the presence of extensive rash with slight constitutional disturbance. Similar points must be borne in mind in diagnosing it from scarlet fever. Throat signs may be of little diagnostic value. In rötheln importance must be attached to the appearance of rash on the cheeks and circumoral area, the presence of distinct maculopapules on the extremities and on the palms and soles, clean tongue, absence of vomiting, and a pulse-rate only increased in proportion to the fever. Mild cases, in which there is little or no rash, are apt to be regarded as due to pharyngeal catarrh with secondary adenitis. Isolated cases should be treated as aberrant scarlet fever or measles, for the sake of precaution.

**Treatment.**—Generally speaking the child should be kept in bed 5 days, indoors 3 days, sent out in the fresh air for 6 days, and then disinfected. The duration of infection must be regarded as possibly 14 days, though it is probably only a week in mild cases. The treatment is purely symptomatic.

## MEASLES.

*Syn. : Morbilli—Rubeola.*

Measles is a very fatal disease and highly infectious in early stages. It plays havoc with school attendance. It may occur from intra-uterine infection and is then mild. Infants are more or less immune, possibly from transmission of immunity from the mother. It is mild during the first 6 months and very severe during the second year of life.



The disease is spread by direct infection; through the medium of doctors, attendants, etc.; or by animals, such as cats. The virus is short-lived outside the body. It is most infective during the invasion period and while the rash is out, especially if there is much catarrh. Catarrhal states and the aggregation of children in schools are potent factors in its spread. Borini (1905) cultivated a small bacillus from the blood. Lesage has isolated a micrococcus.

The *incubation period* is 8-14 days, commonly 9-12 days. Balme (1904) gives a wider limit of 6-18 days. It is often associated with a notable loss of weight, beginning on the third to fifth day (*Meunier's sign*), although the child appears in perfect health. Another early sign is a polynuclear leucocytosis which reaches its maximum about the sixth day before the rash appears. It then falls so much that during the eruption in uncomplicated cases leucopenia is present. Malaise, sneezing and vomiting have been occasionally noted.

**Symptoms.**—It assumes anomalous forms less frequently than other acute exanthemata. Cases may be very mild, with short and slight fever and no complications; of medium severity; or severe, with high fever, long duration, and bad complications. The typical course is an incubation period of 10-11 days, an invasion of 2-4 days, rash for 3-5 days, and convalescence 7 days; in all a total of 3-4 weeks. In the weak and delicate the invasion period may be prolonged to 8 or 9 days.

The attack is ushered in with fever, malaise, cough, and catarrh of the nose, pharynx, larynx, bronchi and conjunctivæ. The discharge from the nose is usually serous, and may become purulent. Epistaxis may occur in severe cases, and is common in the malignant ones. Coryza may be absent, but the absence of all prodromata is rare. Running from the nose, sneezing, swelling of the eyelids, lachrymation and photophobia are frequently present. Vomiting may occur on the first day, on the appearance of the rash, or in the intervening period. After the first day it is due to the catarrh of the fauces or an enanthem in the bronchial tubes. Diarrhœa is fairly common and, if associated with abdominal pain, is a sign that the attack is severe. The cough is short, dry, even paroxysmal, and sometimes barking. It is due to irritation of the fauces, larynx or bronchi. An atypical onset may be characterised by fever and cough, alone or with some submaxillary adenitis. The temperature may then fall but the cough and catarrh persist, and gradually the fever returns. Catarrh of the nose and eyes may not begin until the fourth day. In some cases the disease is ushered in by acute laryngitis so severe to necessitate tracheotomy. Laryngitis generally indicates severity, though it may occur in mild cases. Usually it is slight and disappears as soon as the rash comes out. The cough is hoarse and croupy, and the voice hoarse. The signs are those of croup and may be mistaken for diphtheria. Headache, nocturnal delirium



and convulsions, in the order named are symptoms of increased severity of the disease.

*Prodromal rashes* are quite common in children on the first or second day, rarely after the onset of catarrh, and often before the appearance of buccal spots. They either disappear or merge into the true rash. Goodall (1907) states that the most common one is a punctate erythema, general or patchy, limited to the trunk. In other cases it takes the form of discrete papules, with blotches and irregular patches of confluent papules ; of isolated macules or papules ; urticaria ; or ill-defined erythematous mottling. The rash is sometimes polymorphic, has no special distribution, but is most apt to appear on the trunk and behind the ears, and does not itch. It has no prognostic value.

*Buccal spots* were described by Flindt in 1884, by Filatow in 1895, and Koplik in 1896. They are almost always present 1-3 days before the rash appears. Usually they appear on the first or second day, and if later the rash is often delayed. They are situated on the buccal mucosa, opposite the lower molar teeth ; and may be found on the labial mucosa, palate, uvula and fauces, and even on the gums and under the tongue. They are seen best in good daylight, concentrated by lens or reflected by a short focus mirror, and may be overlooked in artificial light. Koplik described them as “irregular, stellate or round, rose-coloured spots, with a bluish-white speck in the middle. The rose spots become confluent, but the specks rarely or never coalesce. They are *raised* and firmly adherent, and they disappear at the height of the rash.” The specks somewhat resemble miliary tubercles as seen in the meninges. At first they appear as tiny hyperæmic areas. The typical specks, much smaller than a pin’s head, appear in the centre in a short time. This reddened base or areola is not always present. Occasionally the spots are quite large. They may be very few or numerous, and may become confluent. The number does not vary with the severity of the attack or the extent of the rash. Their duration is 2-4 days, and they often disappear with great rapidity. The central speck is the pathognomonic feature and is formed of the puffy apex of a papilla.

Buccal spots may simulate thrush and aphthous stomatitis, but the patches in these affections are larger, more coalescent, more irregularly distributed, opaquely white, and have no red areolæ. The orifices of buccal glands, bubbles of air, or tiny particles of milk, food and inspissated mucus, may cause confusion. In infants, under 6 months, the spots are seldom present until the rash comes out. If they are absent or few in number, the attack is usually mild. They are not found in other conditions.

The *palate* generally shows a blotchy discoloration ; dark or purple patches, triangular or oval in shape and best seen on the hard palate. They usually come out a day before the rash and are least marked, or absent, in mild cases. This is called the “*morbillary enanthem*.” An *erythematous*



*pultaceous stomatitis* (Combe) may precede, by 2 days, or accompany the rash. The mucous membrane of the cheeks and gums is swollen and red, at times violet. The gums are covered with an opalescent or whitish epithelial coating, thin and easily detachable, but they do not bleed or fungate. The fauces and soft palate are injected. Petechial hæmorrhages in the mouth at an early stage indicate severity. They may develop, with ulceration of the mucosa, in the course of the disease.

The post-cervical and, more frequently, the submaxillary glands are often enlarged before the rash appears. Occasionally there is found a general glandular enlargement and splenic hyperplasia. Troublesome cough, dyspnœa, and dry bronchitic râles may also be present, and subside as the rash comes out.

The *invasion* period is terminated by the eruption of the rash on the fourth day, or any day from the second to the ninth. If the incubation period is shortened, the invasion is often prolonged, and vice versâ. The rash generally appears on the fourteenth day after exposure, and is associated with a maximum degree of catarrh. Delay in its appearance is usually a sign of severity. The symptoms increase in severity with the appearance and extent of the rash and include anorexia, thirst, swelling of the face, dulness, drowsiness, delirium, convulsions, and all those due to catarrh. A systolic murmur and slight cardiac dilatation are frequently present during the acme of the rash. Albumin is present for a short time, and the urine gives the diazo-reaction, even 24-48 hours before the eruption.

The *rash* appears first on the forehead, at the margin of the scalp, and behind the ears. Not infrequently it comes out primarily on the buttocks and round the anus. Spreading from the forehead it affects the temples, chin and face, neck, upper arms, trunk, forearms and hands, thighs, legs and feet. It disappears in the same order. It varies between a type like that of German measles or scarlet fever, and one like urticaria. In colour it varies from pale to bright red; pale in mild cases, and livid in severe ones or if there are heart or lung complications. It is composed of maculopapules of variable size, never quite circular, and usually with ill-defined edges. The spots may coalesce into somewhat crescentic patches, leaving intervening areas of healthy skin and causing a mottled appearance. It attains its full development in 3-5 days. Subsequent desquamation is slight or absent. When present, it is branny or of small flakes, and most marked on the trunk, while the hands and feet remain free. Pigmentation may be visible for 2 weeks.

The *temperature* rises as the rash comes out. Frequently it reaches 104° F. at the onset, and falls to normal the next day, remaining down for 1-3 days. It then gradually rises and reaches its maximum with full development of the rash, after which it rapidly falls by crisis. Thus the temperature chart shows 2 marked pinnacles with an intervening period or



remission. Frequently the early signs of measles and fever are present for 12-24 hours; then, in another 24 hours, the temperature falls and the symptoms disappear, and the child seems quite well. Such remissions last for 12 hours to 4 days; sometimes there are more than one but they are then shorter. The signs recur as the rash comes out. In some cases there is no initial rise; while in others the disease runs an afebrile course, notably so in breast-fed babies.

**Varieties.**—Apyrexial measles is a mild attack. Abortive measles (*morbilli sine morbillis*), or measles without eruption, is occasionally seen during epidemics. It is characterised by prodromal symptoms, prodromal rash and buccal spots. In many supposed cases the rash is present, though trivial and overlooked, but in some the most careful examination fails to reveal any sign of it. Fever, coryza, conjunctivitis, etc. may be present. Such cases are usually mild but have proved fatal. *Rubeola sine catarrho* of Willan occasionally occurs and is characterised by rash without catarrh, conjunctivitis or fever. In *black measles* the rash is purpuric, either in consequence of malignancy, or of heart or lung complications.

The *gastro-intestinal type* is one in which the predominant symptoms are diarrhoea and, less often, bilious vomiting. These symptoms come on about the second or third day of the rash, sometimes later, and occasionally in the invasion period. There is often a slight fall of temperature. The stools are frequent, green and foetid; and rarely muco-purulent and hæmorrhagic from necrotic or gangrenous ulceration of the colon. It may terminate fatally in the typhoid state or from broncho-pneumonia.

In *septic measles* there is marked prostration and high fever. It is due to secondary streptococcal infection of the blood and is often fatal in a few days. *Pulmonary measles* usually occurs in the first year of life, and is generally fatal. The rash is more or less livid and fades quickly. There is generally cyanosis with dyspnoea, restlessness and collapse. Acute necrosis of the lung occurs. In *hæmorrhagic measles* the rash is brownish livid in colour, the temperature is high and the constitutional symptoms are severe.

**Diagnosis.**—The most important differential features are the prodromal rashes, remission of temperature, buccal spots, conjunctivitis and early laryngitis, and the diazo-reaction. The urine gives this reaction in measles, but not in German measles. Conjunctivitis begins and is most marked over the bulbar conjunctiva, at the usual site of phlyctenules. This early distribution and limitation may distinguish it from simple conjunctivitis. The leucocytosis during incubation, and subsequent leucopenia, may be of some assistance. In the early stages the illness is apt to be mistaken for acute coryza; and, later on, the skin eruption may simulate that of German measles, serum disease, and various skin rashes. In rare instances the disease has been coincident with scarlet fever (p. 925).



**Complications.**—The pulmonary complications are the chief important ones, and of these bronchitis and broncho-pneumonia are the most frequent, occurring in 10-25 per cent. *Laryngitis tardive*, the form secondary to eruption, is more serious than the primary variety and begins on the third or fourth day of the rash. It may be complicated by microbial infections such as diphtheria, but the diphtheria bacillus is not always found in the membrane. It gives rise to obstruction and glottic spasm, and may produce subsequent ulceration of the vocal cords and arytenoids and perichondritis. Broncho-pneumonia is due to a mixed infection, to virulent streptococci of septic type, or to staphylococcal infection and general pyæmia. It may be followed by acute necrotic pneumonia or bronchiectasis. Sometimes the pneumonia is lobar in type, because of its extent. Atelectasis occurs in babies. Empyema is an occasional sequel. Circulatory complications are mainly dilatation and sometimes myocarditis ; or bradycardia with œdema, albuminuria and syncopal attacks.

Stomatitis is present in 15 per cent. ; diarrhœa in 10 per cent. and often associated with severe abdominal pain and vomiting. Ulcerative stomatitis, cancrum oris, tonsillitis, and parotitis occasionally occur.

The skin may exhibit impetigo, ecthyma, diaper rash, eczema, malaria and herpes. Noma vulvæ is an occasional sequel, and dry gangrene of the prepuce has been reported. The eyes may show chronic conjunctivitis, blepharitis, hordeolum, palpebral abscess, phlyctenules, or haziness and sometimes ulceration of the cornea. Otorrhœa and otitis (p. 995) are common, if adenoids are present, and may terminate in mastoiditis, sinus thrombosis, and meningitis. Excoriations of the nose and lips, rhinorrhœa and epistaxis occur.

The nervous complications are hyperpyrexia ; convulsions ; cerebral symptoms, such as restlessness and irritability, perhaps following a fit ; and encephalitis. Cases of acute encephalitis of various parts of the cerebrum and cerebellum ; false disseminated sclerosis, perhaps a sequel of encephalitis ; acute disseminated myelitis ; ascending paralysis, resembling diphtheritic palsy ; paralysis of the soft palate, independent of diphtheria ; paralysis of the external rectus muscle of the eye ; and peroneal neuritis and atrophy, have all been reported. Other rare complications are severe pains in the joints, purulent arthritis, acute nephritis, parotitis, and empyema of the frontal sinus. The submaxillary and cervical glands are frequently enlarged. Sometimes all the glands are affected on account of the skin changes and toxæmia, and there may be a general tendency to lymphatic hyperplasia. The bronchial glands seem particularly prone to enlargement and secondary tuberculous infection.

**Course and Prognosis.**—Recovery rapidly takes place after the fall of temperature. Prolonged cases may show intermittent, remittent, or continuous fever. The general indications of severity are a short incubation period, laryngitis at the onset, vomiting during the course, diarrhœa and



abdominal pain, epistaxis, livid rash and cerebral symptoms. The outlook is more favourable when the rash develops rapidly, and if it is large and blotchy. A fine rash or one that fades rapidly may be a bad sign. If there are many buccal spots there is usually much rash. Severe nervous symptoms are usually temporary, but a prolonged high temperature or a further rise indicates complications. Debility, rickets, and bronchopneumonia increase the gravity of the case.

The *mortality* is small, except in infants from 6 months to 2 years old, and greatly depends upon the treatment adopted. The disease is seldom fatal within a week. Apparently it is mild under 6 months of age, and especially severe in the second year. In the first 3 years of life the mortality is about 10 per cent., possibly as much as 20 per cent. In the next 2 years it falls to 5 per cent., and from 5-10 years of age it is only 1 per cent. Over that age it is practically never fatal.

Sometimes a *relapse* or second rash develops in 12-14 days, with sickness and fever. The rash is uniformly distributed and typically morbilliform. If the temperature does not come down to normal, the case should be rather regarded as one of recrudescence. Second attacks are rare and usually mild. There may be no rash, except buccal spots, or merely a fleeting eruption. The accuracy of the diagnosis is confirmed by the infection of other children. Many such cases are due to error in diagnosis of the first or second outbreak.

Children who have recently had measles are more liable to whooping cough, influenza, diphtheritic infection of the skin and mucous membranes, and tuberculosis.

**Treatment.**—The child must be kept in bed until the temperature has been down for at least 3 days, and indoors for 3 weeks unless it is warm weather. The ordinary nursing precautions and treatment for fever must be adopted. Keep the room at 60° F. and well ventilated. On account of the eye irritation it should be darkened, or the cot screened from light and draughts. Use boric acid lotion, 2 per cent., for the eyes and borated vaseline for the nose. The mouth and nostrils must be kept clean, the face and hands washed daily, and the body sponged between warm blankets. Hot drinks and a hot bath bring out the rash. The *diet* must consist of suitably diluted milk and carbohydrate foods, and no fruit should be allowed until the risk of diarrhoea is over. The pulmonary catarrh is treated on the ordinary lines of bronchitis, if it is severe. Other treatment is symptomatic and directed to the relief of nervous symptoms, cardiac weakness and severe dyspnoea. Dyspnoea generally depends on dilatation of the right side of the heart. During desquamation a warm bath is given daily and the skin oiled with eucalyptol or carbolic oil, 1 in 40. Good food, cod-liver oil and iron, and general care are very important during convalescence.



*The Prevention of Measles.*—Children should be excluded from schools until 5 years of age. Over this age 75 per cent. of London school children are protected, and the mortality is small among those who contract the disease. Prompt notification is of value, provided that a class is closed for 16 days as soon as the first case appears. If an epidemic is prevalent, every school child with cough, sneezing or redness of the eyes, should be sent home. All the children should be examined daily for buccal spots. Contacts should be quarantined for 16 days, and kept from school for 21 days.

Cases must be well isolated and put under medical care. Other children must be excluded from the house. No child should be allowed to mix with others for a month after the onset of the disease.

**Erythema Infectiosum.**—*Syn. : Fifth Disease—E. infectiosum morbilliforme—E. simplex marginatum—Megalerythema epidemicum.*—Escherich claimed this as a new exanthem in 1896. It is a feebly contagious disease with slight subjective symptoms and a maculo-papular, rose-red rash, most marked on the cheeks, legs and outer surface of the arms. Epidemics occur in families and schools, most often in spring or summer. The common age is 4-14 years. The incubation period is 6-14 days. The rash is usually the first sign. It may be preceded by slight malaise, sore-throat and debility. It only affects the skin, and comes out first and characteristically on the face.

The cheeks present a symmetrical, confluent, rose-red efflorescence, with defined, slightly raised edges or shading off into normal skin, rather sharply limited by the naso-labial folds and the temples, and extending to the angles of the jaw. The skin is hot and swollen, and does not itch. Its colour suggests erysipelas and disappears on pressure. Discrete spots are often seen on the forehead, chin, and about the ears. On the second day the rash appears on the body, chiefly the extensor surface of the limbs and spreading peripherally, the trunk being almost free. On the limbs it is more morbilliform and less rose-red. In many cases it has a lace-work aspect. About the elbows it becomes confluent. On the buttocks it is marked. It is more macular than papular, and shows little elevation except on the face. It disappears from the face in 4-5 days and from elsewhere in 6-12 days. The colour fades from the centre first and leaves red irregular rings visible for a time, the skin assuming a "geographical map" or "marbled" aspect (*cutis marmorata*). Subjective symptoms and complications are rare. Perhaps there is slight fever for 24 hours, and a tongue furred but never like that of scarlatina. There is no coryza, cough, conjunctivitis, glandular enlargement, desquamation or pigmentatton.

It is apparently perfectly harmless, but leads to unnecessary isolation of the child and expense to the parents if mistaken for scarlet fever or measles. The child should be kept in bed while feverish and isolated until the rash has gone.



## CHAPTER LXVIII.

### SPECIFIC FEVERS.

*Varicella—Vaccinia—Variola.*

**VARICELLA.**—*Syn. : Chicken pox—Glass pox.*—This is an infectious disease characterised by the eruption of a papulo-vesicular rash and slight constitutional disturbance. It is sometimes so severe, especially in adults, as to simulate small pox. One attack produces almost complete immunity. It may occur at any age and has been transmitted to the foetus in utero. It is infectious from the onset of the rash and while it is out. Epidemics are sometimes extensive. The contagium has not been isolated although it is present in the pocks, for the disease has been inoculated from them with difficulty. Probably it is conveyed aërially and at times by intermediaries. The virus is easily destroyed under favourable conditions.

The *incubation period* is from 14-17 days. Most cases begin on the fifteenth day ; a few on the thirteenth or not until the nineteenth day.

**Symptoms.**—*Prodromal symptoms* are slight or absent. Occasionally there is a scarlatiniform, purpuric or morbilliform rash which fades soon after the pocks appear ; sometimes a little fever, headache and general malaise ; epistaxis not infrequently ; and perhaps colic, vomiting, and pain in the joints and back. Often the rash is the first sign. The *temperature* rises to 101-102° F., rarely to 104-105° F. It is synchronous with the rash, highest as the rash reaches its maximum, proportionate to its severity, and falls as it fades. Its duration varies from a few hours to 5 days, or it may be prolonged by sepsis. The constitutional symptoms depend on the degree of fever. Delirium is exceptional. Epistaxis may be due to vesicles in the nose.

The *rash* is superficial and comes out on the first day, within 24 hours of the onset. It appears first on, and may be limited to, the trunk. Occasionally it begins on the face and spreads to the trunk and extremities ; or on the scalp, face and trunk simultaneously. It is generally most profuse on the hairy parts and those covered up. It is marked on the trunk, notably the back, and usually spares the hands and forearms. On the extremities it may be limited to a few shotty papules. Occasionally it is vesicular from the onset, looking like drops of clear water on the skin, without any evidence of local inflammation. Most commonly it starts as discrete, small macules or papules with a surrounding red areola. Some papules abort or persist, while most of them become vesicular, forming clear vesicles on a red



base which is never papular. The vesicles are very thin-walled, and vary in size from a pin head to  $\frac{1}{4}$  in. or more in diameter. They are formed within a few hours and attain their full development in 6-24 hours, being then as large as those of variola at the end of 4-5 days. They are generally unilocular and easily evacuated, but evacuation is not a reliable diagnostic feature. They are circular on the scalp and limbs, irregularly round on the face, and elliptical, oval or circular on the trunk. At first their contents are translucent, then somewhat sero-purulent. In 2 days they dry up in the centre, becoming somewhat umbilicated, and form scabs. Both primary and secondary umbilication may occur. The scabs dry up and fall off in 1-3 weeks, leaving an occasional scar.

The rash comes out in successive crops, every 3 or 4 days, and is therefore found in all stages. In number the pocks vary from 1 or 2 to a copious rash all over the body. In marked cases they are present on the scalp, the mucous membrane of the mouth, and even the conjunctival and vulvar mucosa. The degree of pustulation depends on the severity of the infection, the constitutional state, scratching and local infection. Sometimes it is as severe as in variola, and the rash may become confluent. Irritation may be intense, especially under clothing exerting local pressure.

*V. sine varicellis* can occur, a prodromal rash alone being present.

*V. bullosa*.—In a few days large bullæ form, even 3-4 ins. diameter. It is not serious. *V. hæmorrhagica* is rare. The vesicles may be few, or multiple and confluent. The bleeding takes place into the tissues at the base of the pocks, into the skin, and perhaps from the mucous membrane of the eyes, mouth or alimentary tract. Fever and toxæmia are present.

*V. gangrenosa* or *gangrenous dermatitis*.—These two conditions are not identical but *v. gangrenosa* is essentially a gangrenous dermatitis. In unhealthy infants, exceptionally in those apparently strong and healthy, the vesicles become pustules, the suppuration extends deeply, superficial scabs form and separate, and leave cleanly cut, punched out ulcers with black gangrenous sloughs at the bottom. Occasionally hæmorrhage occurs into the vesicles and the base is much inflamed. These ulcers and gangrenous spots may coalesce, involve a large area, extend to the fascia and muscles, give rise to secondary adenitis and abscess, and usually end fatally. The process may be limited to a few spots. A similar affection may follow vaccination, echthyma and pemphigus. In varicella these ulcers are most common on the chest, in vaccinia on the arms, and in echthyma or the buttocks.

**Complications and Sequels.**—Scarring is generally trivial. A few scars are left by an attack of medium severity, usually on the face after a severe pustular rash. Other complications depend on the rash and secondary infection, viz., slowly healing sores, furuncles, abscess, osteomyelitis, pyæmia, suppurative phlebitis, pericarditis, pleurisy, otitis, gangrene of the legs, and erysipelas; retention of urine, due to a pock in the urethra



and swelling of the prepuce (Coombs, 1905); laryngitis and tracheitis (varicellar croup), broncho-pneumonia; stomatitis, tonsillitis, conjunctivitis and vulvitis, and secondary infections of these regions; encephalitis and its sequels; serous or suppurative arthritis; and even cerebral hæmorrhage, ascribed to a pyæmic embolus in a boy 13 years old (Maitland Thompson, 1898). Keratitis, perforation of the soft palate, and Raynaud's disease have been recorded as sequels. Nephritis, latent, slight or severe, is sometimes a late complication, coming on in 15-21 days.

**Diagnosis.**—The age of the patient, presence of vaccination scars, successive crops of rash, characteristic vesicles and surrounding areolæ, and the rash on the scalp and mucous membranes are the chief diagnostic features. From variola it is distinguished by the mild or absent prodromal period, mild character, the distribution, early appearance and rapid vesiculation of the rash, unilocular vesicles, absence of umbilication except during crusting, and polymorphism, several stages of the rash being present on a limited area of skin. A severe attack may be more like variola than modified small pox is. In urticaria there may be transparent vesicles, the *prurigo varicelliforme* of Hutchinson, but they are situated on a papular base, there is no fever, and the itching is intense. Pemphigoid bullæ are much larger and sero-sanguineous. Varicella may assume a pemphigoid form in the newborn. The vesicular rash of congenital syphilis is associated with squamæ and other signs. The scarlatiniform prodromal rash is unaccompanied by sore-throat and vomiting of scarlatina. These diseases may be coincident, but are very unlikely to be simultaneous in onset.

**Prognosis** is excellent. The disease is often so mild as not to require treatment. Fatal cases without complications have been reported in a healthy girl, 8 months old, from severe and extensive rash (Nisbet, 1894), and in a child (Fürbringer, 1896). A purpuric prodromal rash, with hæmorrhages from the mucous membranes, is likely to prove fatal.

**Treatment.**—Keep the nails short to prevent scratching. For its relief order lukewarm baths, vinegar and water, dusting powder, carbolised oil or vaseline, or ichthyol 1 in 50 of vaseline. Use iodoform for gangrenous cases. Attend to the mouth and genitals. Adopt the usual measures in the treatment of fever and isolate the child until the crusts have dropped off.

## VACCINIA.

*Syn.:* Vaccination—Cow-pox.

The inoculation of cow-pox, attenuated small-pox, produces fever, general malaise and a local eruption of pocks, an affection known as *vaccinia*. "Vaccination" is the process of inoculation. Vaccinia is not



infectious but is directly communicable by inoculation. A mother or nurse may develop a vaccine pock on the lip, chin, cheek, occipital region of scalp, hand, mammæ, etc., through direct infection by the contents of a vaccine pustule or from infected towels, etc. Accidental vaccination almost invariably produces a single pock, which may be mistaken for a chancre or malignant pustule. It causes adenitis and leaves a scar.

Vaccination protects absolutely from variola for a period of time varying with the extent of the vaccination and the degree of immunity produced. It is essential in all countries where small-pox exists. Efficient vaccination in infancy, combined with re-vaccination about puberty and 10 years later, will completely protect most individuals throughout life. On exposure to infection, as in the case of doctors and nurses, re-vaccination is advisable if 5 years have elapsed since the last efficient vaccination. In countries where vaccination and re-vaccination are strictly enforced, small-pox is practically non-existent. In countries, where sentimental objections to inoculation and so-called interference with the rights of the subject are allowed undue weight, as in Great Britain, outbreaks of the disease are not infrequent. The risks are those incidental to any scratch, together with those special to the lymph which may perchance be contaminated.

*Vaccine* is lymph prepared from healthy calves under strict precautions to prevent contamination. It is supplied in tubes or on ivory points, and is glycerinated to inhibit and eliminate non-spore-bearing organisms. The use of calf lymph obviates the trivial risk of conveying congenital syphilis, when using human lymph. Erysipelas and other pyogenic infections were said to be spread by human lymph. Unfortunately some of these risks are still present. The fear of tuberculosis can be disregarded, for the calf is killed to see that it is healthy. Yet calf lymph has been known to spread tetanus, and cannot be guaranteed free from pus-producing organisms. Considerable pustulation is often produced, perhaps due to the presence of staphylococci in the lymph, the skin, or on unclean instruments, etc. Further, it is impossible to estimate accurately the strength of the vaccine, as it cannot be standardised, and the same vaccine produces different effects on different individuals.

*Age for Vaccination.*—It must be done before the age of 6 months, unless a medical certificate of postponement or an exemption order is obtained. In Germany the limit is 2 years. The objection to a late age limit for compulsory vaccination is the difficulty in tracing the children. The earlier the operation is done, the fewer are the constitutional symptoms and the less the discomfort of the baby. In the first week of life it is borne best of all. During the first 2 months there is little upset. After 6 months the disturbance is more marked and the fever often considerable. The drawback to early vaccination is that every subsequent rash is ascribed



to it. The rashes of congenital syphilis, intertrigo and eczema etc. are apt to develop about 3-6 weeks of age. Moreover, malnutrition from bad feeding becomes evident about the third or fourth month of life. Hence, if a baby is delicate or ill-nourished, if the skin suggests a liability to disease, or if there is a suspicion of congenital syphilis, it is advisable to postpone the operation. As a rule, it should be done about the end of the first month, provided the child is well and gaining weight satisfactorily. If postponed, it should be done when the weather is reasonably cool and when the child is not teething.

**Mode of Operation.**—Choose the left arm at the level of the insertion of the deltoid muscle, or the left leg about the middle of the outer half of the calf. There is no objection to the latter site in infants, if it is protected from becoming soiled by excreta. Wash the skin thoroughly with soap and water, and dry by rubbing vigorously with a clean towel. Expel the vaccine gently on to the skin, using an india-rubber bulb attachment to the tube in preference to the mouth, and insert it by scratching obliquely with a clean needle. For each insertion make a few downward and transverse scratches, sufficiently deep to cause the appearance of blood, while keeping the skin stretched by means of the hand which holds the arm. A total area of about half a square inch should be inoculated. Three insertions are enough, if of suitable size. A great deal of trouble arises from inefficient vaccination. Public vaccinators make 3-5 insertions, and competing general practitioners tempt the public by offering “one-mark” vaccination. To this there is no objection if it is done thoroughly, but, if only a small area of skin is inoculated, the protection afforded is insufficient and vaccination as a protective measure is brought into disrepute. It is best to make 3 or 4 insertions, each about as big as a 3d. piece, rather than one large one. Large areas produce large pustules and large sores, if the scabs get rubbed off or infected. A similar result ensues from the coalescence of smaller areas too close together. The size of the scar is not a reliable measure of the degree of protection, for it may depend on suppuration.

During the operation the child cries from the restraint of movement, rather than from actual pain. As soon as the lymph is dry, a pad of protective antiseptic gauze is strapped on. This is not essential if the babe is properly nursed and looked after, but it is a safeguard against infection. Clean linen is a cool and satisfactory dressing; wet, greasy and adherent dressings favour rupture of the pustules. The drawback to protective dressings is that considerable suppuration may take place underneath without attracting attention. The skin must not be washed for 12 hours after vaccination.

**Course and Symptoms.**—The scratch heals quickly. On the third to fifth day the site become red and swollen. One or more vesicles appear, coalesce, are fully formed by the seventh to ninth day, and have a swollen



red areola. The areolæ of neighbouring pocks often coalesce and the umbilicated vesicles stand out on a swollen red base. The main vesicles are sometimes surrounded by smaller ones. The corresponding lymph nodes are enlarged and tender. In 2-3 more days the inflammatory process subsides and the vesicles gradually dry up into scabs, or burst and discharge a sero-purulent fluid. The swelling subsides, scabs form and drop off in 1-3 weeks (3-4 weeks from the onset), and reddish scars are left which become bluish-white, white, and finally foveated or honeycombed and depressed.

In severe cases the swelling is extensive, perhaps down to the elbow or involving the whole limb, and there is much pain and constitutional disturbance, viz. fever, restlessness, sleeplessness, anorexia and malaise. The pustule rapidly enlarges and the local inflammation spreads deeply into the subcutaneous tissues, forming an ulcer of variable depth which takes several weeks to heal. All the ulcers may coalesce or black sloughs may form at the bottom of each. The worst cases are those which are the most septic, not the best vaccinated and by no means the best protected.

The fever develops with the appearance of the vesicle, about the third to the fifth day, and attains its maximum of 100-101° F., occasionally 105° F., with the maturation of the pock about the ninth or tenth day, then slowly subsiding. It usually lasts from three to five days, and may be absent. The axillary glands are often enlarged.

*Insusceptibility* is rare. Failures are due to inert lymph, inefficient operation, the production of too much bleeding which washes away the vaccine, washing the skin after inoculation, and occasionally to anæmia or the incubation of a specific fever. No child can be regarded as insusceptible unless there have been three complete failures with different brands of lymph. An interval of a month should elapse between each vaccination for even inert lymph may cause a certain local immunity. Inert lymph may produce bullæ which leave no scars, instead of pocks (*vaccinia pemphigoides*). Partial failures are due to similar causes as complete ones. One or more inefficient pocks are produced and cause further attempts to fail. Such a child is imperfectly protected and should be re-vaccinated at 6-7 years of age.

In rare instances the latent period lasts for 10-16 days, after which the vaccinia runs a normal course. This is probably due to defective lymph. Washbourn (1902) reported a case of *latent vaccinia*, the pocks coming out during scarlatina 2 years after vaccination in which only one place took and that very slightly. Latency has also been recorded of 2 months duration (Brierly), 4-5 months (Metall), one year (G. Harley), and 14 years (Sir T. Watson).

**Complications.**—Extra vesicles, bullæ, ulcers with indurated bases, and gangrene have been noted as local abnormalities. Suppuration, ulceration and lymphangitis, with secondary adenitis and perhaps abscess, are due to



pyogenic organisms in the lymph, and infection at operation or subsequent to rupture of the pock. Vesicles are liable to get rubbed, broken and infected. The local process may end in gangrenous dermatitis (*v. gangrenosa*). Suppurative adenitis is not uncommon. Pyæmia is not unknown. Tetanus is usually due to contaminated lymph, probably through the glycerine. Occasionally, like erysipelas, it results from secondary infection.

*Secondary vaccinia* arises from rupture of the pocks and accidental infection. It is possible up to the ninth or tenth day. Multiple pocks are produced down the arm and forearm, and sometimes on other parts of the body. *Generalised vaccinia* is due to blood infection or auto-inoculation. It begins on the eighth day. The rash is symmetrical, scattered all over the body, and may resemble variola or vary in character, suggesting erythemata, urticaria or measles. Occasionally a lichen comes out in crops of small, red, conical papules about the end of the first week, and may become vesicular or even pustular. Possibly some of these rashes are toxic or co-incident, or are analogous to serum rash. If there is pyogenic infection and suppuration, the usual sequels of these may arise. In one instance encephalitis, ending in microcephalic idiocy, appeared due to this cause. A few cases of nephritis are on record.

**The Effect on other Diseases.**—Vaccination during the incubation period of variola runs a typical course and renders it milder. The paroxysms of pertussis disappear as the pocks develop. Vaccinia seems to render bronchitis and broncho-pneumonia less severe. It does not increase the virulence of scarlatina or measles, but may be delayed until defervescence, and may become confluent and sloughing. It is sometimes decidedly beneficial to existing rashes, does not make eczematous conditions worse, yet is supposed to excite dormant eczema and impetigo. Its effect on gastro-enteric affections is unfavourable. Small nævi can be cured by vaccinating them.

**Prognosis.**—In healthy children normal vaccination produces no ill effects. It does not impair vitality or contribute to mortality. Death is rare and almost invariably due to erysipelas, cellulitis or pyæmia from dirt or neglect. I have never seen syphilitic or tuberculous infection of the wound. Keloid and hypertrophic scars are uncommon. The mortality is higher among unvaccinated than vaccinated babies, probably because debility leads to postponement.

**Treatment.**—Attend to local and general cleanliness, and keep the bowels open. Protect the pocks from injury. Wire shields are unsafe for they are often dirty, are handed on from child to child, and by keeping the parts moist and warm favour rupture. For local inflammation powder freely, apply lead lotion or cold water compresses every  $\frac{1}{4}$  hour, and keep the limb at rest. In ordinary cases dust with boric acid before or when the pocks burst, and use antiseptic fomentations and iodoform for suppuration and ulceration.



## VARIOLA.

*Syn. : Smallpox.*

This affection differs little in unvaccinated children from the disease in adults and is rare, except when there is an epidemic. The nature of the virus in vaccinia and variola is uncertain. According to Klein and Copeman it is a spore-bearing bipolar bacillus. A parasite in the form of spores, sporidium or amoeba, encysted parasite and morula body, has been described. Guarnieri gave these bodies the name *Cytoryctes variolæ*. In vaccinia they do not reach as full development. According to Funk of Brussels they are present in human and glycerinated calf lymph, and in the skin during the incubation stage. They must be sought in hanging drop preparations.

*Congenital variola* does not vary directly in severity with the virulence in the mother. The pustular stage is of the same character as after birth but the pustules are usually larger. Apparently the course may be much prolonged. It has been said to have been transmitted by an infected father, the mother showing no signs of the disease, but this is improbable.

*Variable Severity.*—The virulence of the disease depends on racial and family resistance; acquired immunity, from intra-uterine infection or the transmission of immunity from the mother to the foetus; age, being most severe under 1 and over 30 and mildest about 10 years of age; and nutrition, the fat having pocks which are large and run a typical course, while in the lean and ill-nourished they often abort. The social status has little influence. Antecedent varicella is supposed to attenuate, and scarlet fever and diphtheria to exaggerate the severity of the attack. Small-pox may be very mild in some unvaccinated children, for there is always a small percentage of insusceptibility. It is mild in the vaccinated in whom immunity is waning. The type varies in different epidemics and is less severe toward the end of an epidemic.

*Variola sine variolis* is not rare in the vaccinated. More often one or two pocks are present. In unmodified cases the number varies from a few hundred to thousands. In *modified variola* there are less than 20. It is often called *varioid*. The *incubation period* is 13 full days and the first symptoms appear on the fourteenth day. It must be regarded as infectious from the onset until the complete separation of the scabs.

**Symptoms.**—In varioid the patient is “out of sorts” or “off his food” for a day before the eruption comes out. Or there may be epigastric pain, vomiting and slight fever for a day. Occasionally there is a prodromal scarlatiniform rash. The eruption comes out on the second day. It is



distributed irregularly in infants, and may be limited to the thighs and buttocks. The rash frequently does not pass beyond the papular stage, and the child may be free from scabs in 14 days.

Severe attacks are ushered in by headache, chilliness or rigors, great backache, nausea, anorexia, vomiting, vertigo, malaise and fever. The vomiting, sacral pain and high temperature are the most characteristic features. Incontinence of urine and fæces is not infrequent. Initial symptoms appear early, are marked, and may be as severe in mild or modified cases as in the unvaccinated. Convulsions, delirium and stupor may be present.

*Prodromal rashes* are probably vasomotor in origin and due to toxæmia. They are rare under 10 years of age, and are scarlatiniform, morbilliform or purpuric. They appear first in the inguinal regions, sometimes in the axillary or submammary. The scarlatiniform or "lobster" rash is a uniform brilliant redness, rarely punctate. It comes out on the second or third day, is partial or general, brick-red in colour and becomes dusky or purplish in the flexures. It is most common in the groins, flanks and axillæ, rare on the neck and chest, and may be present on the face. It is of bad prognosis and may be associated with puffiness of the face, subconjunctival ecchymoses, multiple hæmorrhages and blood blisters. A less common and unimportant type is a punctate rash with little erythema. The *purpuric rash* takes the form of small, pin-head petechiæ, usually on the lower part of the abdomen.

The true *rash* appears on the third or fourth day and is preceded by invisible, shotty, subcutaneous nodules. It consists of small red papules and is most marked on the face, arms and legs, especially the backs of the wrists where it feels like shotty papules. It comes out first on the chin or forehead, is more profuse on the back than the chest or abdomen, and appears on the palate and mucous membranes. The papules are large and become vesicular. The vesicles are round or pea shaped, not oval like many varicella vesicles, and take 4-5 days for full development. They are multilocular, umbilicated, filled with clear serum, and do not collapse on puncture. They may become dome shaped when pustular. By the eighth day the rash is distinctly pustular and then the pocks enlarge, each being situated on a swollen inflamed base with a deep red areola. In another 3 days the contents of the pocks are discharged and scabbing commences.

Thus the average sequence of events is eruption of papules on the third day, vesiculation on the sixth, pustulation on the ninth, and the commencement of scabbing on the twelfth day.

The *temperature* rises at the onset, and falls on the appearance of the rash, often to normal when the rash is fully out, but only to about 100° F. if it is confluent. It rises again as the contents of the vesicles become purulent (*maturation fever*), and then slowly subsides in about another



week. Possibly suppuration depends on pyogenic infection and can be prevented by appropriate treatment. During the stage of maturation, or secondary fever, the constitutional symptoms return and are proportionate to the severity of the attack.

For complications and sequels reference must be made to the usual text-books on medicine.

**Diagnosis.**—It is of the utmost importance to distinguish between chicken pox and modified small pox. At the onset it may be mistaken for influenza, typhus fever, scarlet fever, measles, varicella or meningitis. Stress must be laid on the suddenness and severity of the onset with headache and backache; the absence of throat symptoms and marked vomiting; and no catarrhal signs or buccal spots. Prodromal rashes are rare, whereas scarlatina and measles are common under 10 years. In measles the polymorphs in the blood reach 75 per cent. and the temperature rises as the rash comes out. In variola the polymorphs fall to 40-50 per cent., and to 15-20 per cent. in hæmorrhagic types, and the temperature falls with the appearance of the rash.

Mild variola closely resembles varicella. Good recent vaccination scars are strong presumptive evidence against variola. In varicella the rash and fever are synchronous, vomiting and sacral pain uncommon, mental symptoms are absent, and the forearms and hands are usually spared. The differences in the description of the rash and its course in the two diseases must be noted. In varioloid the rash is present in more than one stage, just as in varicella, many papules never become vesicles, others are vesicular in 1-3 days, and umbilication is slight or absent. Hence the difference in these two affections is extremely small and, if small pox is prevalent, it is advisable to regard every doubtful case as variola, to vaccinate the child, and to isolate it or send it to a small-pox hospital.

**Prognosis.**—The severity depends on the efficiency and recency of vaccination. General prodromal rashes are of bad omen. Local ones may be present in trivial cases. Eruption on the second day indicates gravity.

**Treatment** is essentially the same as in adults. The application of pure carbolic acid with a camel's hair brush to the vesicles will abort them. Xylol, m. 10-30, given three times daily in wine or milk, is said to prevent the rash coming out fully and so stop the smell and disfiguration. Of these remedies I have no experience. Ordinary cases of modified small-pox are treated on the same lines as those of varicella. The bed and body linen must be changed daily and the body sponged with tepid water. Cold compresses may be applied to the face and hands if the rash is copious. Or the skin may be dabbed freely with carbolic oil, 1 in 40. This also softens and assists the removal of scabs in later stages. They should be removed early, by poulticing if necessary, should pus be forming underneath. The rest of the treatment is symptomatic.



## CHAPTER LXIX.

### PERTUSSIS.

*Syn. : Whooping cough—Chin Cough.*

Whooping cough, an infectious disease, is characterised by catarrhal congestion of the larynx and trachea and a typical paroxysmal cough. It may prove fatal apart from complications or may leave permanent physical, mental or nervous defects. It is quite as serious as scarlet fever. De Baillou (1578) described an epidemic which occurred in Paris in 1573 and called it *La Quinta* or *Tussis Quintana*. Willis (1667) described it under the name of “chin cough,” possibly a corruption of “Quintana” meaning a 5-hourly cough. It occurs in all countries and in epidemics, being less frequent and less fatal in warm climates.

**Bacteriology.**—Various organisms are present in the sputum. Sprengler and Eppendorf (1897) found a gram-negative bacillus very closely resembling Pfeiffer’s influenza bacillus morphologically and culturally. Jochmann and Krause, Wollstein, Davis, Reyler, and Albrecht have found a similar organism. According to Albrecht it was invariably present in 200 fatal and 70 non-fatal cases. Hensel and Czaplewski found an allied gram-negative bacillus. One of the experimenters developed whooping cough during the investigation and large numbers of bacilli were found in the sputum. They could predict whooping cough from the presence of the organism before the whoop developed. On the other hand the serum of patients during the attack and in convalescence did not agglutinate this bacillus. Bordet and Gengou (1906) obtained a pure culture of a very small, ovoid, aerobic bacillus, gram-negative and non-sporing. It was obtained from the viscid pellet coughed up, and subcultured from the bare places on the medium between colonies of extraneous organisms, for it grows very slowly. It was most abundant on the fifth day of the disease. The serum of children recovering from pertussis had a marked agglutinating effect on the organism while that of healthy children had none. Klimenko, a Russian, claims that he has obtained this organism from the sputum of 5 early cases in children, and produced the disease in puppies by inoculation. Taken as a whole the evidence is in favour of a gram-negative bacillus closely allied to the influenza bacillus.

**Pathology.**—The causation of the whoop is somewhat doubtful. Probably there is a specific laryngo-tracheitis. It is a general infectious disease which starts in the larynx and produces its characteristic symptoms



partly through the local conditions and partly through toxins. According to observations made by von Herff on himself, a small pellet of mucus can be seen on the posterior wall of the larynx at the level of the glottis during a paroxysm, and the removal of this stops the paroxysm. Irritation of this part induced a coughing fit, whereas other parts could be touched with impunity. On examination with a spatula a paroxysmal cough may be caused from reflex irritation of the larynx, even though a whoop has not been noted before. These facts indicate that the spasmodic cough is due to local irritation of the larynx or pharynx, and is somewhat similar to the cough induced by a crumb going the wrong way. According to other theories it is due to inflammation of the vagus, superior laryngeal or phrenic nerve; compression of nerves by tracheo-bronchial structures; increased excitability of the vagus nerve system; a spasmodic bronchitis; a general inflammation of the larynx; or a mixed disease of catarrh and neuritis.

**Etiology.**—It may occur in the newborn, even at the very moment of birth (Sir Thomas Watson), and is not rare under 6 months. During the suckling period infants are less liable because they are less exposed to infection. It is much more common in the first than in the second 5 years of life, and is infrequent afterwards. About 80 per cent. are under 5 years old and half of these under 2 years. Females are somewhat more liable than males. The seasonal incidence depends on conditions likely to induce catarrh. It is most common in spring and autumn, and most fatal in winter. It is more fatal in urban and mining centres than in country districts. The predisposing factors are all those which give rise to catarrh of the air passages, notably measles with which it may be coincident. The *incubation period* varies from 3-15 days and is usually 2 weeks, sometimes it may be as long as 3 weeks. The cough may begin a week before the whoop.

It is *infectious* by contact from the very commencement of the catarrhal stage and before the whoop is recognised. A very short contact is necessary. The infective agent is present in the sputa and is spread by coughing. It may be spread by a third person, through secretions adhering to the clothes, and possibly by the domestic cat. Probably it is only contagious during the catarrhal stage and the child may be free from infection as soon as the whoop has developed. Thus, it very rarely spreads in hospitals from cases in which there are serious complications. Perhaps this is due to the patient being in bed and the infection localised. In practice it is advisable to regard it as contagious as long as there is expectoration and to isolate the child for 6 weeks from the onset. The virus remains active after drying, for epidemics have broken out on ships some weeks after leaving land. The presence of a whoop does not necessarily indicate whooping cough or that the child is infectious. One attack produces, almost invariably, complete immunity. Secondary attacks are very rare and mild,



but it is by no means uncommon for a child to whoop again during a subsequent attack of bronchitis.

**Symptoms.**—It may be divided into three stages, catarrhal, spasmodic, and the period of convalescence; an artificial distinction not maintained clinically. Occasionally in very young infants it begins as acute laryngitis. Usually there is a stage of catarrh for 1-2 weeks before the typical cough develops. The early signs may be erythema of the fauces, sneezing, respiratory catarrh, cough, conjunctivitis and slight fever. Often there are no abnormal signs in the pharynx and chest. Sometimes whooping is present from the beginning. More commonly the cough becomes gradually paroxysmal. It is usually afebrile in its course but preceded by a mild fever and leucocytosis. The *blood* almost invariably shows leucocytosis, varying with the intensity of the course of the disease. This begins with the catarrh, generally before the whoop, and is at its maximum in the paroxysmal stage. It is greatest in the very young and increased by complications. There is no eosinophilia. According to Churchill (1906) it is a lymphocytosis. Lumbar puncture shows an excess of mononuclears.

*A typical attack.*—The chest is first filled with air and then, or without this preliminary process, a series of violent expiratory efforts are followed by a noisy crowing inspiration. The fits of coughing recur and are followed by another and more prolonged inspiratory whoop. The child then brings up some thick, glairy, viscid mucus, and often vomits. In severe cases there is a third bout of coughing and whooping before the expulsion of mucus and vomiting. During bad attacks the chest assumes the position of complete expiration; the eyes are injected, full of tears and protrude; the face is swollen and livid; the tongue projects and its edges are curved upward; the lips are dusky to purple in colour; and the general appearance borders on asphyxia. There may be involuntary micturition or defæcation.

An attack is often preceded by nervous disturbance. The child seems frightened, runs to its mother or nurse, or stops playing and seizes hold of some support. Paroxysms arise spontaneously or are induced by emotion, excitement, and any kind of reflex irritation. They are sometimes preceded by tickling in the throat, a feeling of compression, suffocation or choking, and anxiety. Deglutition, spasm and salivation have been noted by Meredith Young to follow severe paroxysms. Saliva is gulped down as quickly as possible for a minute or two and the stomach becomes distended with air. Paroxysms last from 1-5 minutes. If there is no complication, the child seems well between the attacks. If the fit is mild, the child goes on playing with its toys as if nothing had happened. In severe attacks he is exhausted, sweats freely, breathes more rapidly, has an accelerated pulse, and lies quiet for a time.

The number of paroxysms and the number of whoops in each is very variable. In a severe case there are hourly attacks, rarely over 20 in 24 hours, though as many as 60 may occur. The total number of whoops



may reach 300-400 daily. In some paroxysms there is no whoop. During subsidence of the disease the whoops often cease before the spasmodic cough. The paroxysms are more frequent during the day, unless they are increased from fatigue at bedtime. They decrease during inflammatory complications with fever. The whoop may disappear in severe broncho-pneumonia or pneumonia.

Babies under 6 months of age do not whoop. They have paroxysms of cough and turn red, or more or less black, in the face. In older children the whoop is absent or abortive, if the glottis is not much narrowed.

*Physical Signs.*—Between the paroxysms, due to distension of blood and lymph vessels in the attacks, we find the face congested and puffy, perhaps bloated, swollen eyelids, puffiness under the eyes, suffusion of the eyes, dilated veins on the head and forehead, and possibly hæmorrhages. Examination of the chest shows catarrhal sounds in the lungs, emphysema, accentuation of the pulmonary second sound, and increased cardiac dulness to the right, unless obscured by emphysema.

**Secondary effects and complications** are mainly mechanical. *Sublingual ulcer* on the frænum is present in 25-35 per cent. It is rare under 1 year of age and most common in the second year. It is usually due to mechanical injury, from scraping of the frænum against the lower incisor teeth during the spasm, but it may occur in the absence of teeth. The ulcer is single, variable in size, and rarely painful. It is not typical of whooping cough and may occur in other varieties of spasmodic cough. It may give rise to severe hæmoptysis.

*Severe vomiting.*—Although vomiting is present as a symptom in at least half the cases, in severe forms it must be regarded as a complication. It leads to refusal of food and malnutrition. Some looseness of the bowels, diarrhœa or ileo-colitis, is fairly common. Involuntary evacuations, rectal prolapse, and inguinal or umbilical hernia, depend on the violence of the cough.

Subconjunctival *hæmorrhage* occurs in 2-3 per cent. and may be associated with hæmorrhage into the eyelids, producing a black eye. It is a very suggestive symptom but not pathognomonic. Hæmorrhage may take place from the nose, mouth, ears, bronchi and lungs; and exceptionally into the skin, substance of the eye and brain.

Post-nasal and laryngeal catarrh are occasionally present. Glottic spasm, with rigidity and cyanosis, may occur. Bronchitis is usually added. As it extends downward it sets up broncho-pneumonia, perhaps from secondary infection. It is a common cause of death in infants. Collapse of the lung is frequent in infants and may be accompanied by rise of temperature. In the paroxysms the lungs are unduly emptied, and the viscid mucus prevents the free entrance of air through the narrowed glottis. Acute emphysema is common even in early stages. Occasional com-



plications are mediastinal, sub-pleural, and subcutaneous emphysema of the upper half of the body, pleurisy, pleural effusion, empyema, hæmorrhage into the substance of the lung, bronchiectasis, and tuberculosis of the lymph nodes and lungs. Sputum is often bloody. The strain of coughing produces dilatation and hypertrophy of the right side of the heart. All the cardiac cavities may be dilated, especially in ill-nourished children, partly from cough and partly from toxæmia and malnutrition of the cardiac muscle. Endocarditis and pericarditis are exceptional.

Nephritis is rare, though albuminuria is not uncommon. The urine is pale yellow, specific gravity 1022-1032, highly acid, and contains much free uric acid. It frequently reduces copper salts. This property is most common in the convulsive stage. It is sometimes due to glucose, a true glycosuria, and more often depends on protein derivatives. It has no clinical significance.

Cerebral complications and sequels are infrequent and varied. They depend on hyperæmia or asphyxia, thrombosis, hæmorrhage, sinus thrombosis, meningitis, and local or general encephalitis. Most of them are due to encephalitis. Only in rare instances are they caused by extra-dural, meningeal, or intra-cerebral hæmorrhage. Minute hæmorrhages may produce small foci of sclerosis.

A mild form of meningitis is characterised by subnormal temperature, slow pulse, unequal pupils, headache and torpor. The cerebrospinal fluid, on lumbar puncture, is under pressure and contains about 80 per cent. of mononuclear cells. Many cerebral complications are ushered in by convulsions, generally multiple and often associated with stupor, rigidity of the neck, and perhaps opisthotonos. Severe general convulsions may be due to cerebral œdema from venous stasis while whooping.

Cerebral palsies usually arise in the convulsive stage from hæmorrhage when the paroxysms are violent. During the stage of decline they are more likely to be due to encephalitis. They are ushered in by fits, which may end in coma and death. Or the unconsciousness passes off and leaves behind hemiplegia, less often diplegia, monoplegia and aphasia, and rarely bulbar symptoms, ophthalmoplegia, and nuclear palsy of the sixth nerve. Transitory blindness depends on acute œdema of the cerebral centres. If the pupillary reflex is present, the lesion is between the corpora quadrigemina and the occipital lobes, and the prospect of recovery is good; if it is absent, the lesion is lower down and the prognosis bad. Should coma be also present, the child dies. Rare complications are double optic neuritis, blindness due to hæmorrhage into the anterior chamber or retinal detachment; otitis media, deafness and deaf-mutism; cancrum oris; gluteal and ischio-rectal abscess; and various psychoses, neuroses and scleroses. The psychoses include delirium, screaming, hallucinations, delusions, apathy, imbecility, and weakness of intellect or memory. Paraplegia and peripheral polyneuritis have occurred.



**Course.**—The catarrhal stage is often very short in infants. During the first year of life the characteristic whooping is frequently absent, and the lack of deep inspiration (apnœa) may induce fatal syncope. Occasionally paroxysmal sneezing ushers in the paroxysm and very rarely replaces it. The paroxysmal stage increases in severity to a maximum, and then gradually decreases in the severity rather than in the number of the attacks. Next, the number of paroxysms falls to 1-3 daily, and finally to 1 every few days, eventually ceasing. During subsidence there is an increase in catarrh and muco-purulent expectoration.

The duration varies from 1-3 months. Mild cases recover in 3-4 weeks, leading to undue credit being given to treatment. A prolonged duration is partly due to habit, and partly to constitutional debility, cold weather, dyspeptic troubles, etc. Relapses depend on mismanagement.

Convalescence is tedious, because of malnutrition, anæmia and cardiac dilatation. The child is susceptible to catarrhal affections, influenza, measles, caseation of thoracic glands and tuberculosis.

**Diagnosis.**—Pertussis can occur without whooping in the very young and in the presence of grave lung complications; and whooping can occur without pertussis. The crow of laryngospasm may be mistaken for a whoop. The diagnosis is based on a history of possible infection, the presence of cough in other members of the household, and the hard, irritable, paroxysmal cough in conjunction with puffiness under the eyes, coryza, sublingual ulcer and vomiting. A paroxysmal cough, without vomiting, may be a sign of influenza. The cough produced by enlarged tonsils and adenoids, or pressure on the trachea or recurrent laryngeal nerve, is similar in character. The spasmodic cough of bronchiectasis is most marked in the morning, and is associated with expectoration, clubbed fingers and certain physical signs. Hæmoptysis is common in pertussis and rare in tuberculous lung disease. In acute tuberculosis the child is much more ill than the physical signs warrant; in pertussis the reverse holds good. Expectoration in childhood is most frequently seen in pertussis and bronchiectasis. In the former it is thick, tenacious and hangs from the lips; in the latter it may be offensive. Stress can be placed on the good health of the child between the paroxysms. The introduction of a spatula may bring on choking, retching, and a typical paroxysm; so, too, tickling the nasal mucosa or the external ear, and pressure on the thyroid cartilage. A hysterical cough is barking in type, ceases during sleep, and does not impair the general health. Vomiting is common in many diseases, but in pertussis it is frequent and usually brought on by cough. The initial nervous symptoms, preceding the paroxysms, are a useful aid to diagnosis in some instances.

**Prognosis.**—In itself the disease is rarely fatal, but death is common from its complications. The outlook depends on the age. Although unusually mild in the first few weeks of life, it is very fatal in the first year,



about 25 per cent. dying of those affected, whereas after that age only 3-7 per cent. die. Two-thirds of the fatal cases are under 1 year old. The number of paroxysms per diem is a measure of the severity of the disease, but the frequency of the cough is no measure of the severity of the paroxysm. The prognosis is worst in the first 6 months of life and in rachitic infants under 2 years of age, because of the liability to pulmonary and cerebral complications. Uncomplicated cases, over 2 years of age, generally recover. Complications increase the gravity. Usually they come on during the first 2 weeks, and after that the prognosis becomes more favourable. The common causes of death are bronchitis and bronchopneumonia, terminating in cardiac failure. This accounts for half the deaths of hospital patients. Others die from anorexia and frequent vomiting, asthenia, general malnutrition, or convulsions due to cyanosis, cerebral hæmorrhage, etc. A few die from syncope during a violent paroxysm, and the right side of the heart is found much distended after death. It is essential to keep a careful watch for evidence of cardiac dilatation.

*Post mortem examination* reveals hyperæmia and congestion of the respiratory mucosa from the posterior nares to the bifurcation of the trachea, especially of the posterior wall of the larynx between the vocal cords and the under surface of the epiglottis. The thoracic glands are enlarged and the right side of the heart dilated. There is more or less emphysema and signs of complications, if any.

**Treatment.**—Prophylaxis consists in isolation for 14 days after exposure to infection, for 6 weeks of an attack, and disinfection of the sputa.

Maintain general health, prevent inanition from vomiting, guard against lung complications, try and reduce the frequency and intensity of the paroxysms, and lessen the duration of the disease. Both the mechanical strain and the toxæmia are enfeebling. Let the supply of fresh, pure, mild air be as liberal as possible in view of climatic conditions. If the weather is suitably mild, open air treatment throughout is most beneficial. The child need not be confined to bed nor to the house, if the weather is mild and dry and the case uncomplicated. Sea air is the best. Avoid dust, draughts, wet, cold and wind. If there is pulmonary mischief keep the child in a warm, even temperature, 55-65° F., protected from draughts and from excitement. Two well ventilated rooms are required. Older children should be encouraged to restrain the cough and to cough with the lungs full. Bed is necessary, if there is fever or debility.

The *diet* must be soft, light, easily digestible, and not irritating to the pharynx. Avoid fermentable foods. If there is much vomiting, give small meals after each paroxysm or feed the child again shortly after the vomiting. Attend to the state of the digestive tract and be sure to avoid nauseous drugs. Keep the bowels open with rhubarb, soda and grey powder, if the tongue is furred.



Permit no thoracic compression by clothes or poultices. An abdominal belt, 4-8 ins. deep, worn day and night, is comforting, prevents vomiting and reduces the paroxysms (Kilmer). It is made of linen or flannel, with an insertion of elastic webbing or stockinette, 2 ins. wide, on each side, and laces up the back. It should be 3 ins. in length less than the girth at the navel and applied moderately tightly. Much care is required in the use of such a bandage in infancy, for compression of the abdomen is apt to include compression of the lower ribs, press up the diaphragm, and interfere with expansion or lead to collapse of the lower portions of the lungs.

*Of the Paroxysm.*—An attack can be cut short by dashing cold water in the face; by inducing vomiting, e.g. with a finger or tickling the fauces with a feather; or by pulling the lower jaw downward and forward (Naegele, 1889). The last method must not be used if there is food in the mouth or œsophagus. It is most successful in older children; and may overcome expiratory spasm in cases without whoop.

*Specific Treatment.*—Leuriaux prepared a serum by inoculating horses with broth cultures of his gram-positive bacillus and gave injections of 5 c.c. to children with good results. Manicatide has also prepared a serum. Several observers have noted that vaccination, during the first or beginning of the second stage, lessens the number and severity of the paroxysms. The symptoms abate as the pocks mature.

*Local Treatment.*—Keep the nose and throat clean with sprays of salt solution, boric acid, sod. bicarb.,  $H_2O_2$  in glycerine and water, or with Dobell's solution. Irrigate if the child is old enough. Or insert into each nostril a portion of ointment containing bismuth salicyl., boric acid, quinine, menthol, or various antiseptics. Syringing the ears night and morning with boric acid solution, and painting the tympanic membrane and external auditory meatus with a solution of cocaine and perchloride of mercury, have been claimed as efficacious measures; so, too, painting the fauces once daily with a solution of cocaine 5 per cent. or hydarg. perchlor. 0.1 per cent.; and the application of resorcin 1-3 per cent. to the glottis or larynx.

For inhalation use oxygen for cyanosis, especially at night; a formalin or cresolin lamp; carbolic acid spray, 1 in 40, twice daily for 20 minutes, 1 yard from the child's head, protecting the eyes by bandaging; iodide of ethyl; carbolic acid 0.5-2.0 per cent., thymol 0.2, benzol 0.1, cresol, cresolin, eucalyptus, terebene, tr. benzoin co., etc. The drawback to these methods is the limitation of fresh air, and the advantages are uncertain.

*Drugs.*—The most useful antispasmodics are assafoetida, belladonna, bromides, phenazone, bromoform, codeine, morphine, heroin, citrophen and fluoroform. A simple cough mixture is often as good as anything. Alkaline carbonates are useful in early stages, and expectorants if there is much bronchitis.



Belladonna and bromides must be given in large doses. Belladonna reduces spasms, checks secretion of mucus, and is a cardiac and respiratory stimulant. It is given in doses sufficient to produce erythematous flushing of the cheeks and marked dilatation of the pupils, e.g. m. 4 at 1 year of age and m. 10 at 5 years. After a variable period it loses its effect. It is advisable to combine pot. citrate as a diuretic with large doses. Sedatives ward off complications by diminishing the number and severity of the paroxysms. A mixture of chloral hydrate, phenazone, and vin. ipecac., bromides and syrup is very useful. Eustace Smith recommends butyl-chloral hydrate gr. 1 every 2-6 hours.

Bromoform is somewhat dangerous. Its high specific gravity causes it to sink to the bottom of the bottle and the final dose is an overdose. From m. 1-4 for each year of life are given 3-4 times daily in pure form on sugar, or in an emulsion; e.g. bromoform q.s., sp. vin. rect. dr. 1, pulv. acaciæ drs. 2, syr. aurant. oz.  $\frac{1}{2}$ , aquæ ad. oz. 4; or bromoform q.s., ol. amygdal. drs. 6, tragacanth dr.  $\frac{1}{2}$ , gum arabic dr. 1, aq. laurocerasi dr. 1, aquæ ad oz. 3. It must be dispensed in dark, glass-stoppered bottles, kept in the dark, and shaken before taken. Duncan's syr. bromoform. co. contains bromoform m. 2, aconite, codeine, tolu etc. per drachm. A poisonous dose of bromoform produces narcosis, slow breathing, soft and intermittent pulse, diminished corneal reflex and an erythematous rash. It is treated by artificial respiration, mustard baths, faradisation, coffee, alcohol, etc.

Phenazone is given in doses of gr. 1 per year of life every 4 hours, sometimes in much larger doses or combined with other drugs. It rarely causes rash in children and does not produce depression. It stops the vomiting and reduces the frequency of the paroxysms. Tussol is a compound of phenazone, so is Duncan's elixir pro pertussi, grs. 2 per drachm. Pertussin is a thyme preparation of little value. Fluoroform is expensive and difficult to obtain. A saturated, 2.0-2.5 per cent., solution in water is colourless, odourless, tasteless and apparently harmless. In doses of drs. 1-8 hourly it is said to reduce the duration of the whoop to 6-18 days. For small infants give m. 1 daily after each paroxysm, and increase by m. 1 daily up to m. 100 per diem.

Quinine gr.  $\frac{1}{6}$  per month of life, or gr.  $1\frac{1}{2}$  per year, up to grs. 6, t.d.s., by mouth or rectum, is sometimes efficacious. Other remedies are codeine, morphine, and heroin hydrochlor. in doses of gr.  $\frac{1}{40}$ - $\frac{1}{12}$ , ac. carbolic gr.  $\frac{1}{16}$ , benzol m. 1, hyoscyamus, conium, valerian, tr. grindelia robusta, cannabis indica, and aristochin, alone or in various combination, t.d.s. at 1 year of age. Antispasmine, a mixture of sod. salicyl. and a compound of sodium and narcine, 5-10 per cent. solution in water, is given in doses of m. 3 under 6 months and m. 5-10 at 6-12 months of age. Creosote and carbolic acid are useful for gastric and intestinal fermentation. Oxygen, strychnia and digitalis, or strophanthus, are necessary for cardiac dilatation,



cyanosis and albuminuria; and often stimulants are essential. The usual methods of treatment for dilatation of the right side of the heart, if present, must be adopted. Minute doses of opium can be given for insomnia and excitement, if there is not much bronchitis. Lumbar puncture relieves convulsions. Cod-liver oil, iron and nux vomica are needed during convalescence, and wise management in guarding against exposure to cold, infection, and cardiac strain.



## CHAPTER LXX.

### MUMPS.

*Syn. : Specific or Contagious Parotitis—Cynanche parotidea.*

Mumps is a specific, highly contagious affection of the salivary glands, chiefly the parotid, with usually slight constitutional symptoms and infrequent complications. It is most common in cold weather. Epidemics are frequent in schools. Infants are remarkably immune and the disease is rare in early childhood. It has been recorded in the newborn, but is most frequent from the fourth to the fourteenth years of life.

It is spread almost always by direct contact, probably through the medium of the breath; possibly by infected articles of clothing, toys, etc.; and exceptionally by intermediaries, such as nurses, doctors, visitors and cats. The infective agent and mode of entry are unknown. Peculiarly shaped micrococci have been found in the blood and urine by Capelan and Charrin, and diplo-streptococci by Michaelis (1897). Probably the infective agent enters the mouth or naso-pharynx, travels up the ducts to the glands, and passes into the circulation. Hence, because of their large ducts, the parotids are particularly susceptible to infection. The ducts become swollen and obstructed, and the glands enlarged by hyperæmia and œdema. All the salivary glands may be involved, sometimes only the submaxillary, more often the parotids alone. The inflammation is peri-glandular and inter-acinous, the glandular epithelium being unaffected. Suppuration may result from a mixed pyogenic infection.

*Infectivity.*—Apparently it is infectious during the prodromal period, for as long as 4 days before the onset, and remains infectious for 2-3 weeks. No patient should be allowed to return to school or mix with others until 3 weeks after the onset and at least a week after the subsidence of all swelling.

The *incubation* period is 14-21 days. Dukes (1899) states that it is 13-25 days, in the majority of cases 17-20 days. In 1906 he reported that 22 out of 23 cases began on the sixteenth to nineteenth day. Cases have occurred as late as the thirty-third day after exposure. *Quarantine* should be maintained for 25 days after exposure, but need not be enforced during the first half of that time.

*Symptoms.*—The prodromal period is indefinite, often absent. Symptoms may exist in the form of irritability, languor, fever, general malaise, and diarrhœa or vomiting for a day or two in bad cases. Usually the first indication is stiffness of the jaws, difficulty in opening the mouth, pain on eating, and pricking in the ear or otalgia. Perhaps there is



dulness of hearing or stiff-neck. Occasionally the onset is acute, with vomiting, high fever, and even a rigor or convulsions. Gastro-intestinal symptoms, possibly due to pancreatitis, may be severe. The pain is dull, aching, and localised in the temporo-maxillary articulation. Like the swelling it is often unilateral. It is not always present, even in cases where the swelling is pronounced, and the child may have no discomfort throughout.

The parotid swelling begins on one side in front of and below the ear, elevating and pushing out the lobe. In shape it is that of the gland. It varies in extent, and may involve almost half the face and extend to the clavicle. The lateral pharyngeal wall and tonsil may be bulged inward. It attains its maximum in 3-4 days and gradually subsides in about the same time. The opposite gland is occasionally affected simultaneously, generally in 2-3 days, and rarely not for 2-3 weeks. One side is almost invariably larger than the other. The swelling feels doughy or elastic. It may be limited to the submaxillary glands. The skin is somewhat tense and shiny, and there is rarely any redness or tenderness on pressure. The facial aspect is altered, especially if both the parotid and submaxillary glands are much swollen. The mouth is dry for it is kept partly open. Salivary secretion is normal, deficient, or rarely excessive. The tongue is furred. Stomatitis and pharyngitis may be present. Sometimes there is deafness. The swelling may cause sufficient venous pressure to induce congestion of the face, headache and delirium.

Fever may be absent. Generally the temperature rises with the swelling, up to 102° F. in mild cases and in severe ones to 104° F., or higher. Its course is irregular, not characteristic, and it comes down to normal in a few days. Usually the illness lasts a few days to a week; longer, if the glands are successively involved or complications arise. Diarrhoea may be pronounced at the onset and throughout. Gastro-intestinal symptoms are marked in some epidemics. The spleen is enlarged in severe cases and the cervical lymph nodes may be engorged.

*The Blood.*—Leucocytosis was absent in 7 cases (Cabot), slight in 15 (Sacquépée, 1902). Both Krestnikow and Pick (1902) found lymphocytosis from the onset. Wile's results (1906), based on 20 cases, yielded a total leucocytosis; a lymphocytosis, as a relative and absolute constant sign from the onset until the subsidence of the swelling, most marked in bilateral cases and in children at puberty; a relative decrease in polymorphs, except in orchitis; and no eosinophilia. He regarded the lymphocytosis as a diagnostic feature between mumps and adenitis, provided the relative lymphocytosis of early life is not forgotten. Average relative percentages in these affections were:—

	Lymphocytes.	Polymorphs.	Eosinophiles.	Basophiles.
Mumps	.. 59·37	38·32	2·28	0·43
Adenitis	.. 35·32	62·75	2·26	0·37



**Complications and Sequels.**—Before the age of puberty these are uncommon. *Orchitis* is exceptional under 12 years of age. It is more frequent in some epidemics, comes on at the end of the first or beginning of the second week, generally about the eighth day, and lasts about a week. It is sometimes ushered in with vomiting, is usually unilateral, and may be followed by testicular atrophy. Occasionally in adults it is unassociated with parotid swelling, begins acutely, and lasts 2-3 weeks. *Oophoritis* (*ovaritis*) and *mammary swelling* have been noted in girls, usually on one side, e.g. 13 out of 33 cases, in 8 of which the menses had appeared (Troitsky, 1906). It may lead to atrophy, parenchymatous or interstitial changes. Occasionally the labium major is swollen. Both oophoritis and orchitis may precede the parotid swelling.

*Meningo-encephalitis* of a mild type is not very rare. It appears to be limited to the region of the medulla and pons, and probably accounts for occasional sequels such as headache, optic neuritis, deafness, facial palsy, and herpes of the fifth nerve. Bradycardia and Kernig's sign are symptoms. It has been verified in adults with bradycardia, headache and fever (Chauffard and Boidin, 1904). An excess of lymphocytes was found in the blood and cerebrospinal fluid. Deafness is unilateral and permanent.

Rare complications are paralysis of accommodation, iritis, nyctalopia, optic atrophy, facial palsy, peripheral neuritis, herpes zoster; labyrinthitis; temporary dementia and loss of memory, convulsions, delirium; endocarditis, myocardial degeneration; acute nephritis, usually hæmorrhagic and benign, beginning during convalescence, occasionally chronic and fatal: and the results of secondary infection, viz. suppuration, sloughing and gangrene of the gland; nephritis, pericarditis and pneumonia. It is uncommon to find complications in young children.

*Pancreatitis.*—The pancreas, or abdominal parotid, is sometimes involved in the metastases of mumps. The connection was noted in 1817 by Schmackpeffer. It is more frequent in adults but may occur in boys, even apart from parotitis.

The attack is usually mild, comes on about the third to sixth day (first to twelfth), lasts 2-7 days, and runs a similar course to that of the parotid swelling. Pain is felt in the epigastrium and left hypochondrium. At first it is dull and deep seated, but it increases in severity and may cause syncope. Usually it is paroxysmal, midway between the navel and ensiform cartilage, and radiates to the back or toward the floating ribs. There is much tenderness on pressure and deep palpation is impossible. The swelling is palpable or obscured by abdominal distension; or may be felt after the tenderness has subsided. Nausea and vomiting are rarely absent. The vomiting varies in degree, and may be intractable and hæmorrhagic. Diarrhœa occurs in half the cases, and blood and fat have been found in the stools. Constipation is present in some. Jaundice is uncommon. Cammidge's reaction, acetone and diacetic acid, and



calcium oxalate crystals (often present in pancreatitis), may be present in the urine. There is no glycosuria. The temperature varies from 100-102° F. or may be normal. The parotid swelling subsides rapidly. Fever, vomiting and pain disappear in succession; and there is no evidence of subsequent atrophy. The complication has proved fatal.

Simple acute pancreatitis may result from injury, catarrh of the gut, duodenal ulceration, or infection of the biliary passages. It may terminate in chronic parenchymatous or interstitial inflammation; or in an abscess discharging through the duct or ending in cyst formation. The general symptoms, often absent for a small portion of the gland may carry on its functions, are (1) tenderness on palpation; (2) colicky pains; (3) steatorrhœa, greyish white stools containing fatty acid crystals, globules of fat and clots of soap; (4) azotorrhœa, undigested muscle fibres in the fæces; (5) diabetes, often with acetonuria. Diabetes can occur in children as a functional pancreatic disorder without histological changes in the gland.

The treatment of acute attacks consists of rest, light diet, local applications of heat or cold, and opium for severe pain. Fat must be omitted in pancreatic affections and the amount of meat reduced, if there is no diabetes.

**Diagnosis.**—Glandular hyperplasia is more apt to be mistaken for mumps than mumps for adenitis, except in the primary submaxillary cases which can be distinguished by the course of the disease. Swollen glands are small, circumscribed, of slow development, and do not extend on to the face. The association of pain and difficulty about the angle of the jaw with a parotid swelling is usually conclusive, especially if there is no redness of the skin or faucial affection. Blood examination is of assistance in doubtful cases. Catheterisation of Steno's duct has been recommended. Parotid fluid is normally free from cells. In mumps it is full of polymorphs, large mononuclears, and some cells peculiar to the salivary glands.

**Prognosis** is excellent. Complications are rare and the course usually very mild. About puberty the disease is more serious than commonly admitted. Relapses are rare; second attacks unknown. A secondary rise of temperature indicates extension or a complication.

**Treatment.**—Keep the child in bed until constitutional symptoms have subsided; for 10 days, if over 9 years of age. Younger children with mild attacks must be kept indoors until the swelling has subsided. Give a saline purge, or calomel and scammony; sod. salicyl., or quinine in effervescence (p. 80); and liquid or semi-solid diet. Apply locally hot fomentations, lin. belladon., methyl salicyl., or a mixture of guiacol 1, lanolin 10, vaseline 10 parts, twice a day and covered with oiled silk (useful also for orchitis). Disinfect the nose, mouth and pharynx.



## CHAPTER LXXI.

### THE EYE.

*Blepharitis—Styes—Conjunctivitis—Phlyctenules—Trachoma—The Lachrymal Apparatus—The Ocular Muscles—The Eyeball—Buphthalmos—Keratomalacia—Nyctalopia—Retinitis pigmentosa—Glioma—Amblyopia.*

The newborn child is particularly liable to infection, because there is no secretion of tears to wash away micro-organisms. The common signs of eye mischief are irritation and rubbing of the eyes, photophobia, blepharospasm, and watery, muco-purulent or purulent secretions, with more or less congestion of the conjunctivæ and swelling of the eyelids. Opacities indicate old corneal ulceration.

*Mode of Examination.*—Use no unavoidable force for the hyperæmia of crying is injurious to an inflamed eye. If necessary, the child is placed on its back, held by the nurse, and the head fixed between the examiner's knees. Photophobia results from irritation of the fifth nerve. If present, the child must be examined with its back to the light after instillation of a few drops of cocaine 2 per cent. solution.

For supposed blindness or amblyopia try the "flash-test," i.e. note if the eyes will follow a bright light reflected from a mirror in a dark room. A negative result at 3 months of age is due to blindness or mental defect.

The pupils are naturally small and do not dilate much, even in the shade. In the blindness of optic atrophy the eyes are wide-open, the pupils large, nystagmus often present, and the expression is wandering. Acuity of vision is tested by simple measures suited to the age of the child; so, too, colour vision, red being the colour first distinguished.

Ophthalmoscopic examination requires considerable skill unless the child is unconscious. Ethyl chloride can be given. The pupil should be fully dilated by homatropine 1, euphthalmine 5, or mydrine 10 per cent. solution, a drop being inserted into each conjunctival sac above the inner canthus. For estimating refraction insert ung. atropinæ, grs. 2-4 ad oz. 1, t.d.s., for 3 days before but not on the day of examination. It paralyzes accommodation and the effects take some days to wear off, interfering with observations on the light reflex and inequalities etc. Mydrine and euphthalmine dilate the pupil quickly and interfere little with accommodation.

Note whether there is nystagmus, squint, normal pupillary reflexes, stigmata of degeneration, or cranial defects, and the state of mental and



physical development. Congenital defects include cysts of the orbit, defects in the eyelids, abnormalities of the eye or portions of the eye, and ocular palsies.

*Shades* should be thick, black, dark green or dark blue in colour, and dull on the inner surface. They should be worn whenever atropine is used or if light irritates the eyes.

**The Eyelids.**—*Coloboma* is rare. It is usually limited to one eyelid or the two lids of one eye, exceptionally affecting all 4 eyelids, and occasionally double. Other malformations are often present.

**Blepharitis.**—*Syn.*: *Ciliary blepharitis*—*Tinea tarsi*.—Marginal or ciliary blepharitis is an inflammation or hyperæmia of the edges of the eyelids, which may progress to ulceration. In the first stage, *B. squamosa*, the lids are red, slightly swollen, and show branny desquamation or perhaps yellow crusts. The skin under the scales is hyperæmic. Eyelashes are easily pulled out but grow again. In the second stage, *B. ulcerosa*, there is more swelling, small ulcers beneath crusts, and diseased hair follicles, and the lashes are stunted, irregular, and come out but do not grow again. It is a suppuration of the hair follicles and sebaceous glands. Ulceration may extend to adjacent skin.

It depends on delicacy, bad hygiene, measles, and microbial or parasitic infection, e.g. pediculi. A mild error of refraction, notably myopic astigmatism, is a predisposing factor. Sometimes the disease is unilateral and due to chronic catarrh of the lachrymal sac. It is often associated with phlyctenules, and occasionally due to eczema. The eyelids on waking are stuck together by tenacious secretion and can only be efficiently opened after bathing with warm water. The lids are red and swollen, and may show pustules, ulcers, cicatrices, and matting of the cilia. In bad and prolonged cases the ulceration may destroy the hair follicles and cause complete loss of the eyelashes. Other sequels are hypertrophy of the lid border (*tylosis*), chronic conjunctivitis, closure of the puncta lachrymalia with epiphora and eczema, ectropion of the lower lid and epiphora, with eventual contraction, entropion, trichiasis and secondary superficial keratitis.

**Treatment.**—Suitable glasses alone may cure mild cases. Remove crusts and scabs, after softening them by prolonged bathing, borated starch poultice, or olive oil followed by an alkaline lotion. Bathe with the alkaline lotion and apply ung. hydrag. amm., grs. 8 ad oz. 1, t.d.s.; or ung. hyd. oxid. flav., grs. 4-8 ad oz. 1, or ung. hyd. nitrat. dil. (B.P.), for an hour every morning along the edges of the lids. If there is pus oozing from a raw surface, paint with silver nitrate 1-2 per cent. or protargol 15 per cent., with the eyes closed, wash with salt solution, and then apply the ointment. The solid stick of silver nitrate is sometimes preferable. If pus can be pressed out of the lachrymal sac, the canaliculus must be slit up. Open pustules daily, and epilate involved cilia and those abnormal in colour,



thickness or direction of growth, and remove scales. Give iron and cod-liver oil, and attend to general hygiene.

**Styes.**—A sty, or *hordeolum*, is a furuncle of the lid margin, beginning in a sebaceous gland of an eyelash and forming a hard swelling. It may cause considerable œdema of the lid and chemosis. If near the outer canthus, the cause of the œdema is not always obvious. The pre-auricular lymph nodes may be enlarged and tender. The boil points on the margin of the lid as a small yellow area from the centre of which a hair protrudes.

Predisposing causes are refractive errors, chiefly hypermetropia or hypermetropic astigmatism. Some children have recurrent attacks on account of a constitutional peculiarity, the so-called strumous diathesis, constipation, general ill-health or a susceptibility to acne. Others appear strong and healthy, and are perhaps overfed. The exciting cause is a staphylococcal infection.

**Treatment.**—Attend to the general health and the state of the alimentary tract. Some children need a mild aperient, tonics, ol. morrhue and liberal diet; others require less food and mild saline purgatives. For immediate relief apply hot compresses of boric acid, lead lotion 1 per cent. or alum 1 per cent., give a purge, and if the sty points pull out the hair. If necessary, incise in the long axis of the lid margin, squeeze out the pus gently, and apply hot fomentations. Another suitable preparation is ac. borici dr. 1, ext. hamamelis liq. drs. 2, aq. ad oz. 4, applied on wool several times daily. For recurrent cases apply ung. hyd. nit. and white vaseline, p.a., or ung. hyd. oxid. flav. 1 and vaseline 2 parts, to the eyelids night and morning. The administration of yeast or grape ferment is sometimes beneficial. Should the frequent recurrence be very troublesome, a vaccine may be tried.

**Meibomian or Tarsal Cyst** is due to blockage of a duct with secretion. It forms a pimple on the eyelid and, if suppurating, points on the conjunctival or skin surface. An incision should be made on the conjunctival surface and the contents gently evacuated by squeezing.

**Conjunctivitis.**—The purulent variety, due to the gonococcus, has been described (p. 137). The membranous type is commonly diphtheritic. Simple catarrh is a frequent complication of a "cold in the head," hay fever, measles, cerebrospinal fever, and many forms of local inflammation, and is due to various micro-organisms. The discharge is watery, mucopurulent or purulent. A unilateral one may be due to a foreign body.

**Bacteriology.**—Take a little secretion on a platinum loop, make a film and stain by Gram's method. The organisms in order of frequency are (1) Koch-Weeks bacillus, decolourised by Gram and like the influenza bacillus; (2) Morax-Axenfeld bacillus, a large, broad bacillus or diplobacillus, gram-negative; (3) Gonococcus; (4) Fraenkel's pneumococcus, not very contagious; (5) Streptococci, staphylococci, Friedlander's



bacillus and b. diphtheriæ, all of which are sometimes normally present without symptoms; (6) B. coli and the meningococcus.

*Muco-purulent Conjunctivitis*, commonly due to the Koch-Weeks bacillus, is very contagious and the cause of school ophthalmia or "pink eye." It is characterised by injection or hyperæmia, followed by chemosis, a discharge containing flakes of muco-pus, catarrhal ulcers of the cornea, and sometimes phlyctenules and iritis. The ulcers are often multiple, along the margins of the cornea, and, if they deepen and extend more or less completely round the cornea, may cause its necrosis. The Koch-Weeks bacillus may set up toxic iritis.

*Angular or Follicular Conjunctivitis*, due to the Morax-Axenfeld bacillus, is chronic in character and causes aching of the eyes, heaviness of the lids, a feeling of sand in the eyes, and glueing together of the lids in the morning. The lids are red and macerated at the canthi, and the discharge is soapy, scanty and not purulent. It is often associated with refractive error. Corneal ulceration is rare.

*Membranous Conjunctivitis* is due to a streptococcus, staphylococcus, pneumococcus, gonococcus or the diphtheria bacillus. The streptococcal type is unilateral or bilateral, of variable severity, with a thin or purulent discharge and sometimes a pellicle or membrane on the conjunctiva. It may cause œdema of the lids, ulceration of the lid margins, chemosis, nasal discharge, adenitis, and a temperature up to 101° F. Infiltration of the cornea, amaurosis, toxic iritis, corneal ulcer, hypopyon, staphyloma and loss of the eye are sequels. These results may occur apart from membrane formation.

A staphylococcal infection produces the same symptoms, complications and results, except that it is less serious and rarely causes staphyloma or loss of the eye. The discharge is usually glairy and watery, rather than purulent. The *S. albus* is milder than *S. aureus* in its effects. In diphtherial infection albuminuria is more frequently present, and the pre-auricular and often other glands are enlarged. Differential diagnosis depends on cultural examination of the discharge.

*Treatment.*—In mild cases apply hourly with a tampon of cotton wool boracic lotion, zn. sulphat. gr. 1 or alum grs. 4 ad oz. 1, and vaseline along the edge of the lids to prevent gumming. Zinc sulph. grs. 2 ad oz. 1 destroys the Koch-Weeks and Morax-Axenfeld bacilli. It can be used 4-6 times daily in the more severe purulent forms, until the flow of pus ceases, first irrigating with normal saline solution or washing freely with absorbent wool dipped in water. After the washing instil a few drops into the inner canthus of hyd. perchlor. gr.  $\frac{1}{16}$ , or acid. boric. grs. 10 and zn. sulph. grs. 2, or sod. biborat. grs. 6 and ac. salicyl. grs. 2, or silver nitrate gr. 1-2, each made up with an ounce of water. In very severe cases, without ulceration of the lids or membrane formation, brush the conjunctiva with silver nitrate 1 per cent. daily, and irrigate every 4-6 hours with



1 in 10,000 perchloride lotion. Or irrigate with normal saline and paint with argyrol 20 per cent., a measure often efficacious if there is membrane. Use boric acid fomentations for ulceration of the lids. For the treatment of gonococcal infection *vide* ophthalmia neonatorum (p. 139).

**Phlyctenular Ophthalmia.**—*Syn. : Phlyctenular conjunctivitis or keratitis — Phlyctenule — Lymphatic or eczematous conjunctivitis.*—A phlyctenule is a minute infiltration, elevation or nodule, single or multiple, composed of cellular elements, situated on the ocular conjunctiva and occasionally on the palpebral, with dilated blood vessels (vascular triangles) radiating outward from it. The common situation is the juncture of the cornea and sclerotic. One on the cornea (*P. keratitis*) is superficial and of a uniform greyish colour. If central, it may leave an opacity, lessening acuity of vision or inducing squint. Occasionally a fascicular ulcer travels across the cornea, healing as it goes but leaving a terminal opacity.

Phlyctenules have been ascribed to malnutrition, defective assimilation and excretion, improper food, e.g. sweets between meals, intestinal toxæmia and bad hygiene. They are most prevalent under 10 years of age and often follow measles. Possibly they represent the local reaction of the tissues to the tuberculous toxin, or are due to infarction of dead or attenuated tubercle bacilli. Giant-cells have been found by Leber and Wintersteiner. Nevertheless it would be rash to assume that there is reliable evidence in favour of a tuberculous foundation.

*Symptoms.*—Discomfort, lachrymation and photophobia are present, and greatest when the phlyctenule is corneal. Blepharospasm and eczema of the lids are secondary effects. The nodule is usually reabsorbed and well in 7-10 days. Recurrence is common and may persist for years. Ulceration, due to infection by pyogenic organism, leads to muco-purulent conjunctivitis and local cicatrization, perhaps corneal perforation or permanent infiltration.

*Treatment.*—Prescribe high bracing or sea air, but not the seaside because of the glare, salt baths, liberal diet with omission of sweets and pastries, general hygiene, and tonics such as ol. morrh., malt, hypophosphites, iodide of iron, iron and arsenic. Attend to the state of the alimentary tract. For mild cases use ung. hyd. ox. flav. dil. (grs. 4 ad oz. 1), together with atropine, grs. 4, if there is photophobia. Boric acid lotion, grs. 10 ad oz. 1, can be used as a cold application to reduce inflammation. Atropine has an antiphlogistic action, besides limiting the movements of the iris and coincident alterations in the curve of the cornea. Instil a few drops of a 1 per cent. solution, if the cornea is involved. Cocaine is added if there is much pain or photophobia. Finely divided calomel, applied with a brush, can be used if iodide is not given internally. Do not apply solid nitrate of silver for it may produce a dense silver deposit in the cornea. If the ulcer is purulent and threatening to perforate, use eserine gr. 1-2 ad



oz. 1, 4-6 times daily. In bad cases apply hot fomentations, t.d.s., for 15-20 minutes. A large, thick shade or tinted glasses should be worn.

For photophobia permit no dark room, no bandages, no lying on the face. Sponge with cold water or plunge the face in cold water; and introduce a speculum and expose the eye to light for 15 minutes. Severe blepharospasm may necessitate shading the eyes from light, or even the division of the outer canthus with scizzors. No anæsthetic is needed. The wound soon heals and leaves no obvious mark. After recovery correct any error of refraction. If there is opacity, massage gently with the yellow oxide ointment for 10 minutes twice daily.

**Trachoma.**—*Syn.:* Granular Lids—*Follicular Conjunctivitis.*—Granular lids are the result of a chronic contagious inflammation which responds readily to treatment. It is acute or chronic. At first it is a follicular conjunctivitis, and later it passes into the condition known as trachoma, granular lids or granular ophthalmia. Mild cases show a few follicles (roundish granulations) and a little conjunctival hyperæmia. In more severe ones there are numerous follicles, a red and opaque conjunctiva, swollen lids, photophobia, and a variable amount of discharge, sometimes muco-purulent. Chronic granules look like sago grains. The sequels are cicatrization leading to entropion, trichiasis etc.; cloudiness of the cornea and impaired vision.

*Treatment.*—In acute stages use the ordinary remedies for conjunctivitis. In the chronic stage paint the lids with cocaine, holding them from the eye. Then crush and empty the follicles by pressure with one thumb nail on the conjunctiva and the other outside the lid. After expression paint with argyrol 20 per cent. or silver nitrate 2 per cent., and in 2-3 minutes brush with adrenalin, 1 in 1000, to prevent the blood and serum washing away the silver salt. A lotion of cyanide of mercury, 1 in 10,000, is ordered for home use, a few drops being allowed to trickle in between the lids when washing the eyes with it. The treatment is repeated every third day and the patient cured in 3 weeks. Other methods are the application of tannic acid grs. 20, glycerine oz. 1, or sulphate of copper grs. 2-3, glycerine oz. 1, or tr. iodi and glycerine p.a., over the inner surface of the lids daily or on alternate days. The child can attend school, if he never touches his eyes without washing his hands in a basin of antiseptic lotion.

**Tuberculosis of the Eyelids.**—Small miliary tubercles, which may caseate, ulcerate and coalesce, occur occasionally, more often on the palpebral than the bulbar conjunctiva. The ulcer is round, oval or saucer-shaped, and not indurated, and causes some discomfort or pain. The eyelids may be red and swollen, the pre-auricular glands enlarged and tender, and the discharge purulent. Such an ulcer may simulate chancre. It is treated by scraping and constitutional measures. Rarer varieties are grey or yellow subconjunctival nodules, often mistaken for trachoma,



and usually arising from the fornices and forming granulomatous masses ; “ cockscomb ” excrescences in the fornices, with swelling of the pre-auricular and parotid lymph nodes ; polypoid tumours and extensions from lupoid growths.

**The Lachrymal Apparatus.**—The puncta lachrymalia may be absent or duplicated. Fissure of the gland or sac is a rare congenital anomaly. Unilateral and bilateral absence of tears have been recorded. “ Bloody ” or sanguineous tears come from the conjunctivæ ; some possibly due to vicarious menstruation.

*Dacryo-adenitis*, inflammation of the gland, may be primary, secondary to a styte, or associated with mumps, influenza, measles or gonorrhœa. It is acute or chronic, and terminates in resolution, suppuration or fistula. The physical signs are tenderness and swelling in the upper and outer part of the orbit, œdema of the upper eyelid, and chemosis of the conjunctiva of the upper fornix.

*Dacryo-cystitis*, inflammation of the sac, is more common and is not rare in babies under 6 months old. In them it is due to an impermeable nasal duct, catarrh (blenorrhœa), or the retention of secretory products, which become mucopurulent from mild pyogenic infection at or shortly after birth. Other cases follow injury, nasal affections, measles, scarlatina, etc.

In babies it is generally bilateral and present from birth. By the time the child comes under observation, it is often unilateral through subsidence of the affection on one side. The physical signs are slight ill-defined fulness at the side of the root of the nose, swollen caruncles, epiphora, and sometimes a speck of muco-pus in the inner canthus. On squeezing the swelling a little serum or muco-pus can be pressed out through the puncta into the conjunctival sac or, occasionally, through the nostril on the same side. An acute attack may cause chilliness, fever, severe local pain, redness and œdema. The local swelling is defined and very tender. Later it may extend and simulate erysipelas.

Many cases get well spontaneously. Others require gentle compression of the sac, twice a day, to evacuate the contents, and perhaps it may be necessary to probe the duct and clear away obstruction. For acute inflammation apply lead and opium lotion, or a leech, and give a purge. Neglected cases may rupture externally and end in fistula, if the nasal duct is impermeable. In older children inflammation and abscess may be syphilitic or tuberculous.

**Tears.**—Normally the secretion is only enough to keep the conjunctiva moist, and there is no epiphora, even if the nasal duct is blocked. Excess is due to emotion, overflow from obstruction of the canaliculi or displacement of the puncta, irritation of the ophthalmic branch of the fifth nerve, foreign body, reflex irritation, and hypersecretion occasionally seen in exophthalmic goitre.



**Extrinsic Ocular Muscles.**—Paralysis depends on congenital defects; injury to the head and cerebral affections, such as meningitis, polioencephalitis inferior, cerebral syphilis and tumour; and toxins, notably those of diphtheria, measles, typhoid and influenza. Polioencephalitis inferior may affect the third, fourth or sixth nerve alone, on one or both sides. In palsy of the third nerve the pupil is dilated, and reacts to light but not to accommodation. Squint is due to paralysis, weakness or congenital defect of the muscle, or visual deficiencies. A convergent squint suggests hypermetropia. Treat the refractive error.

*Complete Ophthalmoplegia* may be hereditary and congenital. Gourfein reported one family in which the father and 5 sons, aged 8 months to 12 years, were affected and 3 daughters unaffected. The man's mother showed a like defect and a sister had convergent squint. The signs were complete ptosis, inability to move the eyeball, rotatory nystagmus, and a certain amount of amblyopia.

*Congenital Ptosis* is usually bilateral and associated with defective upward and downward movements of the eyes, weak lateral and good convergent ones. Sometimes the oblique muscles are inert. There is no affection of the intrinsic muscles and no association with other congenital defects. It depends on imperfect development of the muscles or nerve nuclei.

**The Eyeball.**—Microphthalmos has been noted as a congenital and familial affection. It was associated with cataract in a man and 4 out of his 8 children, and in a woman and all her 4 children (A. Bronner, 1902). A translucent globule in the hinder part of the vitreous humour is regarded by S. Stephenson as a common defect. Lang and Collins have described "little rounded bodies of a steel-grey colour which appear to be fluid-containing cysts attached to the optic nerve." They are situated in the fundus in the immediate neighbourhood of the optic disc, and possibly have some connection with the embryonic hyaloid artery of the sheath. No change has been noted in them as age advances. The iris may be absent on one or both sides, or show a coloboma, which may involve the choroid and optic disc, on one or both sides. Anterior staphyloma, often bilateral, is due to failure of development of the anterior chamber or infection via the amniotic fluid. It is decidedly rare.

*The lens* may be congenitally dislocated, as a familial defect. Congenital cataract, of a partial and stationary type, has been recorded by Nettleship and Ogilvie (1906) in 20 members in 4 generations of one family. The sight was little affected. Lamellar or zonular cataract is not infrequently associated with enamel defects in the permanent teeth and a history of infantile fits. It is not due to mercury, for it may be present without such a past history.

**Congenital Glaucoma, or Buphthalmos,** may be present at birth or develop very early in life, and in several members of a family. One or



both eyes look unusually large or "ox-eyed," and the child is said to have "a fine pair of eyes." It is the result of increased ocular tension from developmental defect or an inflammation of the iris and adjoining parts, probably of syphilitic origin. Evidence of congenital syphilis is often obtained. The increased tension leads to progressive uniform enlargement of the whole eye, owing to its distensibility in infancy. The cornea is increased in size, altered in curvature, and cloudy. The anterior chamber is deepened. The optic disc is abnormally cupped and atrophic, the visual fields contracted, and the sight defective. The iris is thinned, stretched, and may be torn. Mild cases show abnormally large eyes, increased tension, a little cloudiness and photophobia. They are liable to hæmorrhage, detachment of the retina, sub-luxation of the lens, escape of the vitreous, corneal ulceration, choroidal degeneration and cataract.

It must be diagnosed from slight congenital opacities of the cornea, interstitial keratitis, and the buphthalmos secondary to ophthalmia neonatorum or severe interstitial keratitis. Some cases get well, with or without treatment. Others end in blindness. The treatment consists in the prolonged use of myotics and mercurials in mild cases. Iridectomy is of great value prior to enlargement, and sclerotomy subsequently.

**The Cornea.**—*Injury at birth* may cause subconjunctival or retinal hæmorrhage, dislocation of the eyeball, retroversion of the lens and vitreous, or traumatic keratitis. The cornea may exhibit at birth an opacity in its anterior layers, with dulness of the surface but no vascularity. The lids and conjunctivæ show some bruising. This is due to injury by forceps or by a contracted pelvis. It may be present in non-forceps cases. A diffuse temporary opacity depends on œdema. A central, oblique or vertical, linear, white scar with an adjacent area of fainter haze, as a permanent sequel, is due to fibrosis secondary to rupture of the posterior elastic lamina of the cornea.

*Keratomalacia* is a softening or gangrene of the cornea, usually bilateral, sometimes found in marasmic infants 3-12 months old. It is identical with "*Hikan*," prevalent in Japan during the diarrhoeal season, and with the *Ophthalmia Braziliana* of South America. It is common in Russia during the great Lenten Fast. It is rare in the breast-fed unless the mother has undergone starvation. In London hospitals it is associated with dangerous marasmus from tuberculosis, congenital syphilis, ileocolitis, broncho-pneumonia and bad feeding. S. Stephenson found the *treponema pallidum* in the scrapings from 4 cases. Probably it can be set up by many different organisms. Infiltration is followed by ulceration, sloughing, sometimes pus in the anterior chamber, and perforation, with extrusion of the contents and collapse of the eyeball. Symptoms are limited to athrepsia, central ulceration and sloughing of the cornea, slight local reaction, and perhaps a little swelling of the lids and blepharospasm. Sensation appears blunted. The prognosis is that of the constitutional



state. About two-thirds of these babies die, generally before the disease has progressed to perforation.

*Family Corneal Degeneration* is very rare. Discrete grey dots or nodules, or a lattice-like system of lines with intervening minute dots, appear in the cornea about puberty. The degeneration is bilateral, central, unassociated with syphilis or inflammation, of slow progress, and seriously impairs sight.

*Treatment of Corneal Affections.*—Use atropine drops, 1 per cent. strength, for abrasions or infiltration; ung. atropinæ for ulceration, with the addition of iodoform, dr. 1 ad oz. 1, if it is septic. In keratomalacia attend to the general health, keep the eyes bandaged up and use the greatest gentleness. Douche frequently with warm boric acid lotion, and instil physostigmine sulphate, gr. 1 ad aquam oz. 1, every 4-6 hours, causing the sloughs to be cast off. For corneal opacities massage with ung. hyd. oz. flav. dil. for 10 minutes, bis die.

**The Iris** is affected with inflammation in the same way as in adults. Sometimes it is tuberculous. Miliary tubercles may be scattered over its surface. They disappear or fuse into a mass of granulation tissue, and may lead to perforation of the corneoscleral juncture and loss of the eye. Occasionally confluent or conglomerate tubercle forms a definite yellowish tumour with smaller surrounding satellites. Or a tuberculous iritis may occur. The ciliary body may be affected primarily by tuberculosis, or secondarily to the iris or choroid. The retina is affected secondarily to the uveal tract or the optic nerve.

**The Choroid.**—Miliary tubercles, up to 70 in number, are found in both eyes, shortly before death in 75 per cent. (Litten) or 82·7 per cent. (Bock), of all cases of general tuberculosis. They vary from pin-point specks to tubercles 2 mm. in diameter. Often there is only one small tubercle, and only in one eye. Solitary tubercles, or more than one, are not infrequent in chronic tuberculosis, possibly in about 10 per cent. Chronic forms exist in which there is an extensive area of inflammation and much granulation tissue; or a single large, conglomerate, circumscribed tumour simulating a glioma. Chronic tuberculosis of the choroid may be associated with tuberculosis of the conjunctiva; may cause retinal detachment; may become obsolescent or re-absorbed, leaving patches like those of albuminuric retinitis, without residual pigment; or may gradually extend and destroy the eye. Tubercle of the choroid is often single and central, whereas gummata are commonly multiple and peripheral.

**Night-Blindness or Nyctalopia, and Day-Blindness or Hemeralopia.**—In night-blindness the child sees well in the daytime, i.e. if the light is good, and badly in the dark or when passing into a shadow. In *hemeralopia* the sight is better by night or in a feeble light, because central vision is impaired and the child is dependent on peripheral vision, seeing better when



the pupils are dilated. It is due to central corneal opacities or, in older patients, to central scotoma, e.g. in tobacco poisoning.

*Night-blindness* may be familial, congenital or acquired; and stationary or progressive. One variety, due to "*torpor retinae*," is common in India and in Russia during the Lenten Fast. It has been ascribed to malnutrition and to deficient alkalinity of the blood. This may be associated with xerosis and conjunctivitis pigmentosa, ulceration of the cornea and keratomalacia. Malannah (1908) states that urotropin is beneficial.

The *Stationary Type* is familial and shows no ophthalmoscopic changes. Possibly there is a deficiency of visual purple in the rods and cones of the retina. Cunier published the history of a remarkable family (600 members, 7 generations), but the nature of the affection was only diagnosed in 1906. Nettleship (1907) studied this family, and his chart embraces 10 generations and 2121 individuals. The disease was traced to an ancestor, Jean Nougaret, born in 1637. It passed from parent to child, was rather more common in females, not due to consanguinity, and was not associated with blindness, other defects or peculiarities. In this family it must be regarded as a dominant, for only the affected transmitted the peculiarity. In other instances it has been transmitted by the unaffected.

The *Progressive Type* is due to retinitis pigmentosa and is associated with progressive atrophy of the optic nerve. It is a slow degeneration or atrophy of the retina, with deposition of black pigment along the blood vessels. Possibly it depends on atrophy of the rods and cones and secondary migration of the pigment. A 4-year old boy had a very granular appearance of the fundus, as if the pigment was escaping from the epithelium. The pigmentation is not always present. Snell (1907) reported it as a familial affection in 12 out of 64 members in 4 generations; 9 males, 3 females. The males transmitted the affection to the daughters and the females to the sons, the other sex being normal in each case. Syphilitic retinitis, or *retino-choroiditis*, may cause night-blindness which progresses for a time and then becomes stationary. It is not always syphilitic and is sometimes associated with microcephalus or cerebral degeneration.

**Glioma** also occurs as a familial disease, e.g. 10 out of 16 children in one family (R. Earle Newton, 1902), 2 being dead and 4 unaffected. The sexes were equal. It was unilateral in 3, bilateral in 7. Six were unoperated on and the eye ruptured in all. A unilateral growth was removed in one case, but death ensued from recurrence at 5 years. The others died under 4 years of age. The prognosis is very bad. Snell (1904) reported 2 cases in one family. One was bilateral, unoperated on and fatal. The other was unilateral, removed, and had not recurred in 6 months. In another case the first eye was removed at 22 months, the second one 2 months later, and the child was alive and well 5 years afterwards. A unilateral case



operated on in the fifth month showed no recurrence at 18 months. A girl under my care had the right eye removed at 16 months, and showed recurrence 8 months later in the form of multiple, rounded, smooth, softish nodules fixed to the skull and not adherent to the scalp. Some became purplish in colour. The left eye was partially proptosed and showed a white growth in the posterior chamber, with the retinal vessels running over it; and the left cheek was white, waxy and swollen. Next the eyelids became purplish. Death ensued 2 months later. Nodules of growth had appeared in the groins. The only hope is immediate removal. It must not be mistaken for a tuberculous tumour.

**Optic Nerve.**—Congenital optic atrophy occurred in 55 members of 16 families (Leber), chiefly but not exclusively in males and transmitted by females. It began usually after puberty, in one patient at 5 years of age. Dimness of vision comes on suddenly and increases rapidly, rarely to complete blindness. Apparently it is a neuritic atrophy and may be induced by toxins, tobacco or congenital syphilis. The nerve may be affected by tuberculosis spreading from the choroid. Miliary tubercles are found in the sheath and septa in general tuberculosis. No special reference, beyond allusions in the text, is necessary concerning the ordinary forms of optic neuritis, choked disc and atrophy.

**Amblyopia.**—The “acute cerebral amaurosis” of William Gay, or “fleeting amaurosis,” is a loss of sight, without ophthalmoscopic changes, due to basal meningitis. Post-eclamptic amaurosis (Ashby and Stephenson) is similar in character, but seeing that it is sometimes associated with aphasia or hemiplegia, it may depend on cerebral changes, the visual cortical centres being involved. Meningitis or encephalitis, with papillitis, is the most probable explanation of bilateral “incomplete post-papillic atrophy of the optic discs; it may be very slight, or associated with squint and nystagmus.” There is usually a past history of fits, headache, vomiting, constipation, retracted head, palsy, squint and unconsciousness, with partial or total blindness which has been more or less recovered from.

**Refractive errors** are described in works on ophthalmology. For 2 hours to 2 days after birth there is frequently 4-7 D. of myopia, with dilated pupils and almost immobile eyeballs (Elshnig, 1906). It disappears after instillation of atropine and is replaced by hypermetropia or emmetropia. Refractive errors, notably astigmatism, are a cause of headache. Myopia is often hereditary, but generally is the result of undue use of the eyes in reading badly printed school-books in an unsuitable light.



## CHAPTER LXXII.

### THE EAR.

*Hearing—Congenital Defects—Affections of the External Ear—Foreign Bodies—Otitis Media—Earache—Otorrhœa—Mastoiditis.*

Perfect hearing is essential to the perfect acquirement of speech, and for intellectual and moral development. Children with defective hearing learn speech slowly, often slur the consonants, and have a somewhat toneless voice. Primarily sounds are registered in the cortical auditory centres and sense memories of them are there stored up. In the next stage the child attempts motor reproductions of these memories (echolalia), and gradually develops intelligent speech.

*Physiology and Anatomy.*—The blood vessels of the auricle are superficial and susceptible to cold. Chilblains and frost-bite are common. The nerve supply is abundant. The auricular branch of the vagus is partly distributed to the external auditory meatus. Hence, local irritation of the meatus may cause “*ear cough*.” The epidermis, lining the auditory canal, contains sebaceous and ceruminous glands and many short hairs, and is continued over the drum as a very thin layer.

The middle ear consists of the tympanum or tympanic cavity, the bony part of the Eustachian tube, and the mastoid antrum. The continuity of these parts is easily made out in infancy and early childhood. The tympanum and Eustachian tube are the remains of the first post-oral visceral cleft, and the antrum is also developed from this cleft. The cavity is a small irregular space between the drum and internal ear. It is lined with ciliated epithelium on its floor and toward the Eustachian tube, through which it communicates with the pharynx. The mucous membrane is closely adherent to the bone, which becomes readily affected in disease. The plate of bone separating the tympanum from the meninges is extremely thin and sometimes congenitally absent. Inflammation may readily spread from one to the other. The petro-squamous suture runs across the roof of the tympanum and is not obliterated until the end of the first year, and not always then. The close relationship of the mastoid cells to the internal ear is also of much importance. During early life the mastoid bone is small and its cells undeveloped. In children the external lamina of this bone is thin and easily perforated.

The Eustachian tube maintains equality of pressure in the tympanum with that of the external atmosphere, and is the channel by which the



secretions of the mucous membrane escape from the middle ear. Obstruction to its orifice or canal induces distension with secretion, indrawn drum, deafness, tinnitus etc. It is nearly horizontal at birth. It opens into the pharynx at the level of the hard palate until the age of 9 months, then a little higher. Though it is wider than in adults, catheterisation is almost impossible, for the fossa of Rosenmuller is nearly imperceptible.

The external auditory canal is almost straight, running a little forward and inward; and the tympanic membrane is placed more horizontally than in adults, being almost horizontal at birth.

*Examination of the Ear and Hearing.*—Enquire into the family history. Seek for evidence of congenital syphilis, a previous attack of scarlatina or measles, etc., nasal or respiratory affections, earache and otorrhœa. Ascertain the mode of onset and duration of symptoms. Note the general condition of the child, its mode of life, diet and environment, and its facial aspect. Examine the throat and nose for enlarged tonsils, adenoids, post-nasal catarrh and nasal obstruction. Syringe away any wax or discharge present.

Apart from dulness of hearing, the ears should be examined in the first year of school life and after exanthemata. The usual tests are whispered speech, ordinary speech, a bell, watch, Galton's whistle and the tuning fork. The whispered voice is heard by the normal ear at a distance of 18-27 feet. The ear should be turned toward the examiner, the other one plugged with wool, and the eyes covered or closed. Cases are classified as bad deafness when the whisper cannot be heard at a distance of over 2 yards; medium, at 2-4 yards; and slight, if heard over 4 yards away. Sibilants, *s* and *sch*, are usually heard badly in disease of the conducting apparatus, *r* in defects of the drum, and *f* in labyrinthine disease. A watch is not a reliable testing instrument, for its tick varies and is a compound of sound and noise. The tuning fork only gives satisfactory results in very intelligent children. If it is not heard in the deaf ear, when applied to the vertex or on the mastoid, there is probably serious disease of the auditory nerve or nucleus, fortunately rare in childhood. For infants and the very deaf reliance must be placed on sudden sounds, e.g. that of a bell with the clapper fixed by a spring to the outer surface, and noting whether the eyes are attracted toward the sound. Care must be taken to eliminate the effects of vibration. It is important to note what escapes the child, quite as much as what he hears. Inability to hear soft sounds, the murmur of the breeze, the shivering of the leaves, and the buzzing or humming of insects, is a serious drawback to the intellectual appreciation of life and nature.

Note the characters and appearance of the pinna and external auditory meatus, and if there is otorrhœa. Affections of the canal can generally be diagnosed without a speculum, by simple inspection in a bright light. For examining the drum use Brunton's otoscope, containing a lens, or a



speculum and reflected light from a forehead or hand mirror. Warm the speculum first. An infant or young child should be held sideways on the nurse's lap with the head against her breast. Under 4 years of age, the canal is straightened by pulling the auricle outward, downward and forward, or downward and backward. Above that age it is pulled upward and backward. Wipe away discharge, if present, with a dry antiseptic mop; and gently syringe out any wax which impedes the view. Look for a relaxed or depressed membrane, inflammation, perforation, and a "dancing bubble," a minute speck which pulsates and is due to perforation.

*Inflation* is used for diagnosis and treatment. In Valsalva's method the patient holds the nose, shuts the mouth and swallows suddenly. If the tube is patent, air rushes up into the tympanum. In Politzer's method a small rubber bag with a soft nozzle is used. The patient takes a mouthful of water, with the nozzle of the bag in one nostril and the nostrils compressed by the finger and thumb, and swallows as the operator compresses the bag. If the child can be taught to puff out the cheeks, swallowing is not always necessary. A rubber tube, with an ear piece at each end, is inserted into the ears of the patient and operator. Complete absence of sound and no improvement in hearing indicate Eustachian obstruction. A "pop" is heard, if the obstruction is relieved; "bubbling," if there is mucus in the tube or fluid in the tympanum; and "whistling," if there is perforation.

Children, especially when young and nervous, must be examined with great care and tenderness. Otherwise they may be hurt or frightened and it is impossible to form accurate conclusions. If there is great tenderness an anæsthetic is necessary. No probe or director should be used in examining for a foreign body, without full illumination. If the drum cannot be seen because of swelling, reduce the swelling and examine later. Catheterisation is rarely advisable.

**Congenital Abnormalities.**—Developmental errors chiefly affect the auricle. It is formed by the coalescence of 6 tubercles on the margin of the first branchial cleft. The ear may be represented by a lump of skin. *Supernumerary auricles* are not uncommon, usually in the form of one or more small tubercles of skin near the lobule of the ear or on the side of the neck. Rarely one is modified to imitate a second pinna. They are due to persistence or modification of the primary tubercles and should be removed. *Inequality* in size results from excessive or defective development of one pinna. A very small ear may be associated with facial hemiatrophy. *Peculiarities* in shape are present in many idiots and are regarded as stigmata of degeneration. *Protruding ears* are due to developmental defects, ill-fitting caps, strings behind the ears, or pressure on the turned-over ear during sleep. A suitable apparatus worn at night cures some cases. Or a plastic operation can be done, a piece of skin being removed from the auriculo-mastoid juncture. Congenital defects of the



pinna are often combined with *occlusion of the canal* and branchial fistula, imperfections in the deeper aural structures, and sometimes with congenital morbus cordis and other malformations. The meatus may be closed by a membranous septum.

**External Ear.**—*Erythema auris*, the result of sunburn, may need a cooling lotion or soothing powder for its relief. *Intertrigo* is a superficial erythema, often due to binding the auricle to the head before it is properly washed and dried or to the use of irritating powders and lotions. Wash the part gently twice a day, dry by dabbing, and dust with emol keleet, or zinc oxide and starch. *Eczema* is common in all its forms, acute and chronic. It is dry, vesicular, papular or pustular, and often secondary to facial eczema or local irritation from otorrhœa. The pustular variety is sometimes of the nature of an impetigo, and called *impetigo auris*, and is due to microbial infection. Chronic eczema leads to thickening and deformity of the pinna, and extends into the meatus, causing swelling and obstruction. Other affections include herpes, erysipelas, gangrene, Raynaud's disease and tuberculous ulceration around ear-rings.

**Treatment.**—For details refer to the section on eczema. Protect the ear from air, water and injury. Do not allow remedies to get to the tympanum. For the cure of a very chronic eczema it may be necessary to set up an acute attack.

**Foreign Bodies** are frequently unsuspected, giving rise to no symptoms. Some are soft, e.g. peas, and tend to swell after insertion. Others are hard, smooth or rough, e.g. beads, stones, slate pencil, fruit stones, etc. Occasionally insects of various kinds or maggots are found. Foreign bodies more often give rise to injurious consequences from the attempts made to remove them than when left alone. Both hearing and life have been sacrificed to unskilful management. In the case of a bead or pebble there is no urgency, for there is normally a tendency to spontaneous extrusion by the growth of the epidermis outward.

Insects can be killed by filling the ear with warm carbolic lotion or a pledget of wool soaked in chloroform. Maggots are killed by carbolic lotion, or a mixture of chloroform 1, sp. vin. rect. 2 parts, and then removed by syringing. For bodies which do not swell, syringe with warm water; for peas and similar soft substances, syringe with warm oil, instilling glycerine in the intervals to induce shrinkage. Always direct the stream against the upper wall of the canal, with the head inclined toward the affected side. Inject the fluid regularly, not in jerks. Do not syringe unless the body has been seen. Therefore, examine first in a good light and do not try to locate it by probing in the dark. Attempts to remove it by forceps are more likely to push it further in. If there is inflammatory swelling reduce it first by antiseptic and sedative lotions before trying to remove the body. If syringing fails, recourse is had to the use of a wire loop for hard bodies and a fine crochet hook for soft ones.



**Impacted Wax.**—The insertion of water into the ear, as in bathing and plunge baths, hardens the wax into a mass which is retained. Another cause is the use of appliances, such as a small sponge on a stick, for cleaning the ears. Wax is protective and only the excess should be removed. Obstruction of the canal and catarrh of the middle or external ear are predisposing factors. Impaction produces dulness of hearing, actual deafness and noises in the ear, but rarely any injury to the drum. The wax is easily removed, for it rarely forms the hard, massive plug found in adults. A plug of compressed scales, due to desquamative dermatitis, is more irregular and lighter in colour. On syringing it swells up and causes increased pressure and headache. It can be removed by forceps, after softening it for 2 days by instillation of ac. salicyl. gr. 5, sp. vin. rect. m. 90, glycerine oz.  $\frac{1}{2}$ . For the removal of wax use a syringe with a fine nozzle, a lotion of sod. bicarb. or borax, good light and a forehead mirror. The wax can be softened by dropping in, 5-6 times daily, a few drops of sod. bicarb. grs. 20, glycerine m. 30, water to oz. 1. The stream from the syringe must be directed toward the roof of the canal. As soon as the wax is evacuated, the hearing returns and is often very acute. Afterwards insert a plug of wool for a few hours to protect from cold. If the hearing is not improved, examine for disease of the middle or internal ear.

**Furunculosis.**—A boil in the canal is very painful and may cause inflammatory swelling over the mastoid. Insert a pledget of wool soaked in ichthyol and glycerine, p.a., with another pledget to keep in the solution without exerting pressure, once or twice daily until the boil dries up. It prevents further infection and eczema of the meatus, if the boil suppurates, bursts or is opened. Should incision be necessary, use a special knife so shaped that the point is directed outward.

**Earache** is a symptom not always easy to diagnose in infants. Reliance must be placed on the troubled face, resting the head on the nurse, refusal to rest it on the affected side, rolling the head on the pillow, rubbing or pulling the ear, putting the hand to the ear or side of the head, and crying on sudden change in position of the head, on touching the ear, and on pressure below the meatus. It is not always due to ear disease, being sometimes reflex, e.g. along the auriculo-temporal nerve from dental caries or during dentition.

Cocaine, followed by large hot fomentations, is the best treatment. Or insert a few drops of (1) warm olive oil and chloroform, p.a. ; (2) cocaine, 2 per cent. in aniline oil and sp. vin. rect. p.a. ; (3) adrenalin 1 in 2000 ; (4) ac. carbolic grs. 5-10, glycerine oz. 1. Never use cold applications and avoid spirituous lotions. Other remedies are irrigation with hot lysol or lysoform lotion, fomentations, mustard leaf over the mastoid, and possibly a few drops of tr. opii internally. Paracentesis is unnecessary and Politzerisation unsuitable.

**Otitis Media.**—Inflammation of the middle ear is of grave importance in early life. During infancy it may cause deaf-mutism or be the starting



point of ear disease at a later age. It is acute or chronic, primary or secondary, and gives rise to earache, fever and subsequent otorrhœa. The discharge is serous, seropurulent or purulent.

*Etiology.*—It is extremely common under 2 years of age and bilateral in 50 per cent. Rasch (1894) found no exudation before the third day of life. Primary cases are exceptional; the cause may be overlooked. Few are due to blood infection. Nearly all are secondary to direct infection, via the Eustachian tube or lymphatics, from the throat, nose and nasopharynx, especially adenoids. Often it complicates an acute catarrhal respiratory or pneumonic affection. Of the infective fevers scarlatina is the great cause, measles next, and less frequently diphtheria, varicella, variola and typhoid fever.

It complicates cerebrospinal and pneumococcal meningitis, influenza, pertussis, various throat affections, stomatitis and other mouth affections. Ill-health and bad hygiene are predisposing causes. It is frequent in infants dying from congenital syphilis, tuberculosis, marasmus and disorders of the alimentary tract.

*Bacteriology.*—The pneumococcus is present in 80-90 per cent. in infancy. Preysing (1906) found it in almost all of 81 cases, 73 bilateral, in 100 children, aged 1 day to 3 years, examined after death. The otitis was pneumococcal in 112 out of 125 positive results.

Miller (1907) examined after death the ears of 50 children under 4 years old, and found 20 bilateral pus, 17 unilateral pus, and 13 normal; the pneumococcus present in nearly all; and that 29 out of the 37 with otitis had broncho-pneumonia. Under 10 years of age this organism predominates, and the younger the child the more probably is it the cause of the disease. Pyogenic organisms are often found in secondary cases and the tubercle bacillus in a few.

*Morbid Anatomy.*—As soon as the mucous membrane undergoes catarrhal inflammation, it appears to lose its bactericidal properties and may become secondarily septic. There are 3 types of otitis, serous, suppurative and necrotic, all of which are seen in scarlatina. The mucosa is swollen and injected, and secretes serum, sero-pus or pus, and becomes thickened by small-celled infiltration. The exudation fills the cavity, causes necrosis and ulceration, and rupture of the drum and otorrhœa. In bad cases the disease produces destruction of the drum, loosening and detachment of the ossicles, periostitis, caries or necrosis of bone, involvement of the internal ear, total deafness, and palsy of the seventh and eighth nerves. The inflammation may extend to the meninges, via the lymphatics or the squamo-petrosal fissure, and cause meningitis; or may set up mastoiditis or labyrinthitis.

*Symptoms.*—Otitis can exist without earache or pain, notably when it complicates acute general disease. Or the expression of pain may be absent on account of the mental state, and even in normal children. In



infants acute pain is constantly absent or unrecognisable, and indeed all signs may be lacking. Usually there is restlessness, insomnia and sharp cries, and rarely mastoid tenderness. Sometimes the pain is agonising, throbbing, and causes incessant shrieking. High fever, coated tongue and delirium are not uncommon; and there may be convulsions, head-retraction, vomiting, and rapid breathing. Meningitis is often suspected in infantile otitis, generally a bilateral and purulent one, because of the head retraction and cerebral symptoms. The drum should be punctured in doubtful cases. The otitis may be an antecedent or a sequel of meningitis (p. 644).

The onset is often insidious, with dulness of hearing or deafness as the only symptom. Subjective noises are difficult to ascertain in the young. Many cases are mild or sub-acute. Possibly the child complains of slight earache and noises in the head, and appears dull of hearing, otorrhœa developing in a few days. These children are generally delicate and have some catarrh of the nose or fauces.

The temperature varies. In mild and subacute cases, and those in the course of acute disease, there may be no fever. Generally the temperature rises abruptly to 101-105° F., and falls sharply as soon as the drum is perforated. The fall is not necessarily simultaneous with the relief of tension. Usually the fever lasts 1-5 days.

Otorrhœa is the confirmatory sign; "an abscess has broken in the head" is the common description. The discharge may be so scanty and tenacious as not to be visible externally. Wipe out the meatus with a pledget of absorbent wool. Insert a smaller pledget for a short distance. If on removal it is moist with mucus, there is a perforation, for mucus is not secreted in the canal.

*Physical Signs.*—If the meatus and canal are swollen, inflamed and tender, it is impossible to make a satisfactory examination. At first the large capillaries of the drum are seen to be congested; next the minute capillaries on the periphery become visible; and finally the whole drum is vividly red or almost purple in colour, and the individual vessels indistinguishable. This vivid redness is not often seen, for desquamation of the epidermis in the form of minute dots begins about the same time. The membrane becomes so dotted with white epidermis that the redness is not visible. The drum in early stages is thickened, œdematous and opaque, from serous exudation, rendering the ossicles eventually invisible. Sometimes it bulges. In infants it is often deficient in lustre, sodden, and does not bulge. The presence of rhinitis in such cases is a suspicious indication.

On perforation discharge is visible and must be removed by mopping, in order to see the deeper structures. A perforation looks like a small drop of muco-pus on the drum, which if wiped away at once re-appears. On inflation by Valsalva's method the drop is enlarged. It is usually



situated above and behind, or in front and below the centre of the drum. At times the whole drum is destroyed, especially in scarlet fever. This may be preceded by membranous deposit on the drum or in the middle ear. The odour is foetid.

**Varieties.**—All the signs and symptoms may be present in a severe form and yet subside spontaneously without perforation or formation of pus. In some of these the pus escapes through the Eustachian tube. In others no pus is found on paracentesis. A non-purulent exudation may become purulent. After a variable period of earache, the temperature rises, pain becomes worse, and the drum is found swollen and oedematous at first, then deep red or purple, and later of a yellowish colour.

*Scarlatinal otitis* varies in frequency with the severity of the throat symptoms, and the presence of adenoids, carious teeth, and septic mouth conditions. It is either due to the specific organism or to the spread of non-specific ones from the naso-pharynx to the tympanum and antrum. It is usually bilateral, though not necessarily simultaneous on the two sides.

An acute catarrhal otitis may occur at the time of the rash or within the first 10 days. The mastoid antrum is only affected secondarily. The perforation, if present, is small and high up in Shrapnell's membrane.

A suppurative variety, due to pyogenic infection of the catarrhal one, commonly begins in the second week, sometimes not until the fourth week or during convalescence, and occasionally in the acute stage. It may give rise to no symptoms, being only recognised when the otorrhœa begins; to earache, deafness and rise of temperature; to increasing delirium and coma; or merely signs of aural discomfort or a rise of temperature. Perforation may result within 24 hours or after a slower course. The perforation is large and in the inferior segment. There is no Eustachian obstruction or tinnitus, and deafness is usually slight unless adenoids are present.

Some attacks pass into acute necrotic inflammation, rapid destruction of the tympanum, separation and discharge of the ossicles, and permanent deafness. The cavity is filled with pus and granulation tissue, perhaps in large masses forming aural polypi which may be even present at the meatus. The discharge is profuse and soon becomes foetid. The seventh and eighth nerves may be involved, and meningitis may supervene.

Infection of the mastoid cells and antrum, an osteitis of the antrum and cells, occurs early. Sometimes there is a primary otorrhœa which subsides and is followed by a secondary offensive discharge, due to accumulation in the antrum. Other sequels are perforation of the labyrinth, caries and necrosis, cerebral and cerebellar abscess, thrombosis of the lateral sinus, permanent deafness and deaf-mutism.

About half the cases recover in 2 weeks to 2 months. Adopt intratympanic treatment for a month and then, if there is no improvement, open the antrum or do a radical operation.



*Typhoidal Otitis* begins in the third to the fifth week with otorrhœa and rarely pain. Occasionally it is acute, unilateral, and may involve the mastoid. The deafness of typhoid is generally of central origin, and sometimes due to catarrh of the fauces and Eustachian tube. The otitis of *measles*, diphtheria and pneumococcal infections has no distinctive features. In measles it generally occurs during desquamation, with rise of temperature and earache, and rarely ends in ulcerative destruction or mastoiditis.

*Influenza* gives rise to hæmorrhagic otitis, primary mastoiditis, or rapid caries or necrosis of the ossicles or mastoid. In this otitis the pain is sudden in onset, neuralgic in character, intermittent, the paroxysms being longer and more frequent at night, and is little or not at all relieved by perforation. Deafness comes on more gradually than in simple otitis, is little relieved by inflation, and lasts for 3 weeks or more. The drum is swollen and intensely congested, showing punctate or diffuse ecchymoses. The tympanic mucosa is much swollen and forms flabby œdematous granulations. The discharge varies in character from scanty sero-sanguineous to profuse purulent fluid. Nervous prostration and insomnia are marked.

*Tuberculosis of the middle ear* is sudden in onset, chronic in course and practically painless. It chiefly affects the poor in large towns. The bacillus reaches the tympanum via the Eustachian tube, or possibly by the blood. The discharge is thin, semi-purulent and very foetid, sometimes thick and curdy, and the bacillus may be found in it. The drum is pale, sodden and œdematous, and perforations have indolent edges. There is a tendency to flabby granulations, and caries of the mastoid and inner tympanic wall. The facial nerve is soon involved. Early operation is the proper treatment.

**Course and Complications.**—An ordinary case of otitis gets well and leaves no permanent effects. Deafness is most likely to follow scarlet fever; or after mild but recurrent attacks. The ossicles may be detached and thrown off. Other complications are caries and necrosis of the walls of the tympanum, mastoiditis, labyrinthitis, sinus thrombosis, meningitis, cerebral abscess, and abscess in the neck. A cervical abscess may be glandular, in the cellular tissue and due to direct infection from diseased bone, or a collection of pus in the jugular vein.

*Chronic Otorrhœa* is not infrequent. Otorrhœa is a sign of suppuration. If it comes from a meatal boil, it is intermittent and lasts for a day or two. If due to a foreign body, it is associated with swelling and may be continuous. A discharge of 6 weeks duration is "chronic," and in 90 per cent. comes from the middle ear, indicating perforation and chronic inflammation of the tympanum, and caries or necrosis of the mastoid. The signs of chronic disease are a constant foetid discharge, exuberant granulations, and caseous foci. The longer its duration, the greater is the probability of bone disease in and around the cavity. It is generally septic or tuberculous.



**Mastoiditis** is primary or secondary. The primary influenzal form gives rise to local, lancinating pain, tenderness over the mastoid antrum, local heat, and possibly fever. It often causes rapid destruction. *Primary caries* of the mastoid, ending in abscess, is not uncommon. The patch of caries is usually along the line of the masto-squamosal suture. It is a slow painless formation, sometimes taking a month, without fever, and produces a swelling behind and perhaps pushing forward the ear, generally extensive destruction of bone, and no intracranial symptoms. There is no history of otorrhœa, no earache, and the membrane is normal. *Secondary mastoiditis* begins 2-8 weeks after tympanic suppuration. It may start acutely with rise of fever, rigors and severe pain.

The signs of mastoiditis in infancy are restlessness, crying, paroxysmal pain which is worse at night, rubbing the occiput, tremor of the lower jaw during pain, fever up to 105° F., cessation of otorrhœa, glandular swelling and torticollis. In older children there is often no fever and no local signs, but the pulse is generally accelerated and pus constantly oozes through the perforation in the drum, after mopping, showing that there is accumulation and retention. Local signs indicate that the inflammation has reached the surface. They include swelling, heat, redness, œdema and tenderness over the antrum or pre-mastoid lamina, if it has started as a periostitis. When due to extension from the tympanum the signs and symptoms are less acute. There may be persistent and deep-seated pain, increased discharge, and bogginess or bulging of the posterior superior wall of the meatus, just external to the drum. The mastoid cells form the outer wall of the lateral sinus. If they are diseased, thrombosis may ensue.

*Treatment.*—Apply leeches or cold. Make an incision down to the bone, or trephine the process if the symptoms persist. A permanent fistula indicates caries. The earlier the operation, the less is its severity, the more rapid the cure, and the smaller the loss of hearing.

**Labyrinthitis** may be primary in mumps, or secondary to tuberculous infection or otitis media. It leads to destruction of the labyrinth and deafness, or deaf-mutism if it is bilateral and occurs in an infant. Loss of bone conduction is the sign of inflammation or destruction of the labyrinth. Occasionally it is due to pressure on the nervous structures and is relieved by perforation of the drum.

**Treatment of Otitis and Otorrhœa.**—Prevention consists in the removal of adenoids and large tonsils, attention to carious teeth, and hygiene of the mouth and nose, especially in exanthemata. The mouth must be kept clean during illness, the throat sprayed and the nose syringed with a mild antiseptic lotion, e.g. ac. carbolic grs. 30, sod. bicarb. grs. 30, water 1 pint, warmed by dilution with hot water.

In acute otitis make use of the measures for the relief of earache (p. 991) and give bromides. Let an infant lie on a large pillow on the nurse's lap, with the affected ear uppermost. It must not be rocked or



jogged up and down. Keep the room dark, cool and quiet. Feed with a spoon, for sucking causes pain. Give liquid diet and a purge. Similar measures are suitable for older children, together with liq. quin. salicyl. m. 10-15. q.i.d. Examine the ear, under anæsthesia if necessary. Try and reduce acute inflammation, so that it does not end in suppuration, by 1-3 leeches on the inner side of the concha, the meatus being plugged with wool, or in front of the ear, or over the mastoid if pain is felt there as well. If an infant is teething, blood may be abstracted by lancing the gums. Other measures are hot instillations or irrigations, hot fomentations or bran bag, etc., a mercurial and saline purge, and vin. antimonialis.

*Paracentesis* does no harm, if the meatus is kept aseptic, and should be done early in scarlatinal cases for they almost always suppurate. Incise the drum if it is red and bulging, if the clinical symptoms are severe, or if rolling the head from side to side suggests labyrinthine trouble. Enlarge a perforation, if it is too small. Cocaine solution 2 per cent. in aniline oil and sp. vin. rect. p.a., 5-10 drops, produces anæsthesia in 5-15 minutes. Aqueous solutions do not penetrate the drum and reach the nerve filaments, which terminate on the inner layer. Any discharge present must be first syringed away. If there is a perforation, the solution causes a burning pain before the cocaine takes effect. It is a mistake to wait for perforation, for the pain and danger are prolonged, and the resulting hole is often inadequate for drainage. The size of the hole is of more importance than its situation. Select a spot below and behind the handle of the malleus, and be sure that the knife is sharp. Keep the hole open until exudation into the middle ear has ceased. Paracentesis is not essential in serous otitis, but may relieve grave symptoms although no pus escapes. Afterwards irrigate with hot boric acid lotion for a few minutes every hour for a few hours, instilling a few drops of carbolised glycerine, 5 per cent., between the irrigations. Cover with a pad of absorbent wool to soak up the discharge.

After 3 weeks, if the discharge continues in spite of free drainage, trephine the mastoid although there may be no evidence of mastoid disease. It is the only means of securing efficient drainage and is recommended for purely mechanical reasons. For pain and profuse discharge in otitis of streptococcal, influenzal or scarlatinal origin, operate in a week, because of the liability to bone disease.

*Chronic otorrhœa* depends on inefficient treatment of acute otitis, debility, tuberculous infection, recurrent infection from the naso-pharynx, and imperfect drainage. Therefore enlarge perforations, get rid of granulations, polypi, epithelial debris and inspissated pus, and treat the nose and naso-pharynx. Use mops or a suction apparatus, e.g. Siegle's pneumatic speculum, for cleaning the meatus. Syringing, politzerisation, and hydrogen peroxide are all liable to drive septic matter further in. The peroxide is very useful for cleansing. Fill the ear until bubbling ceases and leave the fluid in for 5 minutes. Wash it away with warm boric lotion and dry by mopping.



As a rule the simplest plan is to illuminate the meatus well, mop it out with a dry aseptic mop of singed wool, and clean it with mops dipped in one of these lotions:—alcohol; biniodide of mercury gr. 1 in sp. vin. rect. oz. 6; bichloride of mercury 1 in 2000; carbolic acid 1 per cent., salicylic acid or boric acid or resorcin 1-2 per cent. in rectified spirits. Spirituous lotions destroy granulations by dehydration, lessen swelling, and are antiseptic. The meatus and concha must be anointed first to prevent smarting and chapping. The discharge usually diminishes in 1-2 weeks. Then, if there is no bone disease, use an astringent lotion of lead acetate or zinc su'phate, grs. 2 in glycerine oz. 1. The insufflation of dry powders is inadvisable as they form hard masses and hinder drainage. For the same reason do not insert wool into the meatus. It also causes maceration and excoriation. A gauze drain along the floor of the meatus up to the drum exerts capillary attraction but may set up eczema. All lotions should have a higher specific gravity than pus, in order to penetrate, and should not be made up with water for water soddens the tissues and promotes the growth of granulations and polypi.

A useful method is to syringe with a boric lotion and dry by mopping; instil freely ac. boric. dr. 1, sp. vin. rect. drs. 2, glycerine ad oz. 1, and leave it in for a few minutes; then suck it out with an eye-dropper with a rubber bulb. Various antiseptics can be used in a similar manner.

Inflation helps to drive out secretion through a perforation. If the hole is large, there is little risk of driving pus into the antrum and in many cases the antrum is already infected. It is better avoided, for it is unnecessary if drainage is efficient, and it gives shocks to inflamed structures and may drive in septic material from the pharynx. If the discharge is very offensive instil daily, or every other day for a week, into the deepest part of the meatus, 2-3 drops of a warm 5 per cent. solution of the double cyanide of zinc and mercury. More radical measures should be entrusted to the hands of a surgeon or specialist on diseases of the ear.



## CHAPTER LXXIII.

### THE SKIN.

*Dermatotherapy—Intertrigo—Erythema—Seborrhœa—Eczema—Psoriasis—Scleroderma—Corns—Warts—Molluscum—Lichen—Alopecia—Sudamina—Strophulus—Acne—Boils—Impetigo—Contagiosa—Ecthyma—Pemphigus—Herpes—Tuberculous Affections—Parasitic Diseases.*

In examining the skin it is essential to inspect the whole surface, though not necessarily at once. Some rashes, e.g. eczema, assume different types in different regions. The examination should be conducted with warm hands in a good light and warm room. Note the general condition of the skin, the quality and texture of the hair, and the distribution of abnormalities. A dry, lustreless appearance shows a tendency to xerodermia and a liability to eczema. Ichthyosis appears first on the shins. Look for moles, warts, hairiness, scratches, and patches of keratosis pilaris and lichen spinulosus, due to hypertrophy of hair follicles. A fleeting erythema is a sign of digestive disturbance. Dermatographia indicates vasomotor trouble.

**Dermatotherapy.**—At birth the skin is very delicate and subjected to a great change in its surroundings. The vernix caseosa is rubbed off. Irritant oil or fat, caustic soap, water of unsuitable temperature, too vigorous drying, and unsuitable clothing may exert their injurious influence at the time of the first bath as well as subsequently. These factors are accentuated by irritant powders, sweating, urine and fæces, unclean diapers and general lack of cleanliness. Preventive measures consist in the care of the skin at birth and the use of baths (p. 72). The special measures include soaps (p. 72), powders, oils, fats, ointments, pastes, glues, plasters, lotions, and baths (p. 75).

*Powders* are used to dry up secretions, cool the skin, and reduce inflammation and swelling. The chief kinds are the starches of rice, wheat, or potato; emol keleet; lycopodium; terra silicea; mag. carb. (calamine), mag. silicat. (talc), zn. oxid., zn. oleat. and bism. subnit. To any one, or mixtures, of these boric acid is often added.

*Oils.*—The chief ones are ol. mor., ol. olivæ, ol. ric., ol. rapæ (cheap) ol. amygdal. dulce (expensive); and the mineral oils, petroleum, paraffin and liquid vaseline. The *Fats* include white vaseline, made from the residue of petroleum, and the more uniform yellow vaseline; and the



animal fats lanoline and adeps lanæ, lard and adeps benzoat., cold cream and ung. rosæ, wax, and ung. cetacei.

*Ointments.*—The basis of an ointment must be non-irritating, not rancid, and of a proper consistency. Cold cream and ung. rosæ are excellent, if fresh, but the water evaporates and they may go rancid, unless benzoin 2 per cent. or carbolic acid 1-2 per cent. is added. The best emollient is fresh pure lard, benzoated to prevent decomposition. Commercial lard is a mixture and unsuitable. Lard, combined with an animal or vegetable oil, penetrates into but not below the skin. Theobroma (cacao) oil is the best.—Lard can be combined with wax and oil; or with oleic acid, for admixture with alkaloids. Vaseline is protective and non-absorbent. Its irritant effects, if any, are due to impurities such as sulphur. It is the proper vehicle for antiseptics, germicides, parasiticides, astringents and counter-irritants. If it is desirable for the active constituent to penetrate and pass through the skin, it must be combined with pure hydrated lanoline, a sebaceous substance which is practically unalterable, never becomes rancid and is unaffected by chemical reagents. Such a base is required for mercury, pot. iod., quinine and narcotics. It is rarely suitable as a base alone, and is often mixed with cacao oil, wax, or vaselin, e.g. lanolin pur. 65, paraffin 30, ceresin 5.

*Lubricants* include mutton tallow, fats, oils, creams, glycerine of starch, and mixtures such as boroglyceride 1, lanolin 2, ung. rosæ 6 parts. As a *soothing ointment* use:—(1) ac. carbol. gr. 5, calamin. gr. 20, zn. oxid. gr. 30, ung. rosæ ad oz. 1; (2) zn. oxid. gr. 15-30, ung. picis dr. 1-2, ung. rosæ ad oz. 1; (3) pulv. camph. gr. 30, zn. oxid. dr. 2, glycer. dr. 2, adip. benzoat. oz. 1-2, cochineal gr. 1, ol. rosæ m. 1.

Ointments should be spread freely on the woolly side of lint and bandaged on; and removed by means of a dull knife or absorbent wool. At first the strength must be mild, for it is impossible to estimate the susceptibility of the skin. The ingredients often act better in combination than separately. A few ointments must be rubbed in vigorously.

*Creams.*—An excellent cold cream, and a suitable basis for an ointment, consists of spermaceti 1, ceræ alb. 1, ol. amygdal. 10, aq. rosæ 10 parts. Carron oil is a mixture of ol. olivæ and aq. calcis p.a., and forms a suitable cream with calamine or zn. oxid. 10 per cent.

*Pastes.*—The dry parts must equal the fats. Useful ones are starch and vaseline p.a.; zn. oxid., starch, vaseline, lanoline, p.a.; zn. oxid., ol. olivæ, p.a. (Lassar's zinc oil); and zn. oxid. 1, starch 1, vaseline 2 parts (Lassar's paste). Pick's paste is a useful excipient for applying drugs without the necessity of bandaging. It is made of tragacanth powder dr. 1, glycerine dr.  $\frac{1}{2}$ -1, aq. rosæ 3 oz.

*Gelatines, Glycogelatines, Zinc gelatines or Glues* are composed of gelatine alb. dr. 2-4, zn. oxid. or calamine dr. 2-3, glycerine dr. 4, water 1 oz.,



liquefied in a glue pot and painted on. Other suitable vehicles for ac. salicyl. and chrysarobin, etc., are collodion and traumaticin (guttapercha dr. 1, chloroform oz. 1).

*Salve Muslins* are made by spreading muslin with lard and wax, lanoline or vaseline, to which is added the drug required, e.g. ichthyol, ac. carbol., ac. salicyl., resorcin, sulphur, etc. It is trimmed to fit the part. A cheaper method is to spread a thick ointment on butter-cloth. Biersdorf's *plaster mulls* contain various medicaments. A *salve stick* is made of cacao butter 4, wax 2, lanolin 1 part.

*Lotions* are cooling, sedative, antiseptic, astringent or stimulant. They are brushed on, or applied on thin gauze to permit evaporation. Simple soothing lotions are warm rice water, lime water, lead lotion, or solutions of boric acid, sod. bicarb. or sod. hypophosph. dr. 1-2 ad water 1 pint. *Calamine lotion* consists of calamine 1, zn. oxid. 1-2, glycerine 4, aq. calcis 8, aq. rosæ 32-64 parts. A mixture of calamine 1, olive oil 4, and lime water 4 parts, is very soothing.

**Erythema and Intertrigo.**—An erythema is a uniform redness of the skin of varied causation, mainly vasomotor or toxic. Erythema due to irritation by the sun (*E. calorum*), lotions, mustard leaves, etc., is the first stage of dermatitis. *E. neonatorum* is the normal redness of the skin at birth. Other varieties are *E. pernio* or chilblain (p. 453), *E. nodosum* (p. 535), *E. infectiosum* (p. 950), and *E. multiforme*.

Erythematous rashes are sometimes caused by belladonna, phenazone, quinine, veronal, resorcin, iodoform or boracic lotion. Drug rashes are often polymorphous. The phenazone rash is scarlatiniform, burning and itching, and may involve the buccal mucosa. There may be fever and subsequent desquamation.

Sometimes it is morbilliform, and generalised, or mainly on the limbs. Occasionally one or more small red, raised, infiltrated patches of "fixed erythema" come out in a few hours and last for a week or more, perhaps leaving a pigmented patch for months.

*E. multiforme toxicum* is due to a toxin or to gastric irritation. The chief causes are dyspepsia, constipation, tainted food, auto-intoxication and infectious disease. It chiefly affects the chest, neck and face. *Septic erythema* and *serum disease* are of toxic origin.

**Intertrigo** is an erythema due to friction between two moist surfaces, especially the folds of the groins, between the genitals and thighs, between the buttocks and round the anus, behind the ears and in the axillæ. It is worst in the "bathing drawers area," and is often called a "napkin rash." If limited to the buttocks and perineum, it may be, but ought not to be, mistaken for the rash of congenital syphilis (p. 871). At first there is a simple erythema. Warmth, moisture and additional irritation lead to the formation of small, superficial vesicles or denudation of the skin over a larger or smaller area, producing intertriginous ulceration. Thin scabs



form and, under suitable treatment, recovery is rapid. The ulceration is most extensive on the buttocks because of the faecal irritation. Malnutrition, combined with lack of cleanliness and failure to keep the parts dry, is the chief cause. Other common causes are the use of diapers which have been dried but not washed, or washed in soda and water but not thoroughly rinsed out in pure water afterwards; and various dusting powders. Overfeeding with starchy foods renders the stools acid and irritating. The rash is much more limited in area than that of syphilis, and differs from eczema in the absence of weeping.

In its treatment it is important to prevent chafing, by means of lint soaked in oil, or covered with zinc ointment, and kept between the opposing surfaces. Before vesiculation or ulceration has occurred wash with warm boric lotion, dry gently and thoroughly, and dust on a powder of ac. salicyl. 3, starch 10, talc 87 parts. In the later stages rely on ointments of zinc, boric acid, zinc and vaseline, or more stimulating ones of resorcin gr. 5 or ichthyol gr. 10 ad oz. 1. Internally give rhubarb and soda, grey powder, calomel or aromatic chalk powder, according to the state of the digestive organs.

**E. Multiforme.**—In its simplest form this consists of a variable patchy, evanescent efflorescence (*E. fugax*) sometimes papular (*E. papulatum*), without heat, pain or swelling, and perhaps itching. An *exudative* variety is sometimes called *E. marginatum*, *circinatum*, *iris*, *gyratum* or *annulare*. It comes out on the backs of the hands and feet, the lower arms and legs, and may affect the mucous membranes. At first it consists of small, vivid, light red spots. These become dusky and fade in the centre, and enlarge peripherally, perhaps coalescing. Sometimes there are small peripheral vesicles (*herpes iris* or *E. vesiculosorum*) or urticarial spots. They fade in 2-6 weeks and leave some pigmentation. Occasionally the rash is associated with slight evening fever, pains in the limbs, articular swelling, especially in knees and ankles, and renal and intestinal hæmorrhage. Such cases are closely allied to purpura hæmorrhagica. It is rarely severe in children. Keep the child in bed, apply cooling powders and ointments, warm baths, and give calomel and aspirin or salicylates internally.

**Seborrhœa.**—The secretion of the sebaceous glands is often excessive, especially on the heads of infants. The newborn may present a patch of seborrhœa capitis.

*Seborrhœa sicca*, pityriasis simplex capitis, scurf or dry scaly seborrhœa, is a branny desquamation composed of dried sebum. It occurs in quite young infants, is very common about puberty, and may simulate tinea tonsurans. Wash the head with soft soap twice a week. Apply nightly a lotion of ac. salicyl. dr. 1, ol. ricini dr. 2, ol. rosæ m. 10, sp. vin. rect. ad oz. 6; or ac. salicyl. gr. 10, sulph. præcip. gr. 5-30, ung. rosæ oz. 1.

*Seborrhœic eczema* is the moist and crusted type. It takes the form of a dirty yellow or brownish yellow patch of dried, greasy secretion on



the scalp. Small patches are simple and uncomplicated. Neglected ones are liable to spread over the whole scalp and set up an eczematous inflammation which extends to the face, neck, trunk and limbs. The nose, mouth and eyes escape. It is most common in overfed, constipated babies and may clear up spontaneously on a mixed diet. There is a superficial catarrh, exudation, scabbing and not much itching. The redness is seen at the edges of the patch, being hidden by the dried secretion in the centre. A well-defined crescentic red edge is visible at the margin of the scalp in extensive cases.

The primary local patch must be soaked with warm oil or a mixture of ac. salicyl. 2-5, ol. ric. 40, ol. oliv. ad 100 parts; or poulticed with a borated starch poultice (p. 77). It can then be removed by washing gently with a superfatted soap and water. The process must be repeated as soon as the scab forms. After cleansing, apply precipitated sulphur gr. 5-30 in an ounce of benzoated lard or of ung. aq. rosæ; or zn. oxid. gr. 10, hydrarg. ammon. gr. 3, lanoline and vaseline aa  $\frac{1}{2}$  oz. For chronic patches use resorcin gr. 5-10 or ichthyol gr. 15-30 in an ounce of a simple excipient or of ung. ac. borici. Keep the head cool, and uncovered indoors.

The general seborrhœic eczema, which affects the whole body in adults and is somewhat like a syphilitic roseola, is rare in children. It simulates rose rash, and is distinguished by the presence of seborrhœa capitis. Its treatment is that of eczema.

**Eczema.**—Eczema is a simple dermatitis of a catarrhal nature. Its first stage of erythematous patches may be absent. In the second stage vesicles or papulo-vesicles form and burst, discharging a clear, yellow, viscid, slightly alkaline, irritant, serous fluid. The discharge stains and stiffens linen, or dries and forms crusts on the skin. The corneous layer exfoliates and leaves cracks or fissures. Usually there is much itching, and in acute cases considerable constitutional disturbance. The secondary effects are due to itching, scratching and infection. Apparently eczema is often a localised œdema of the skin with the formation of deep-seated vesicles in the rete mucosum, non-microbic and therefore due to a toxic state of the blood.

**Etiology.**—Dryness of the skin and excessive sebum are predisposing causes. It is as absurd to rigidly maintain that the disease is purely constitutional as it is to believe it invariably a local affection of the skin which can be cured by local measures alone. In the majority of cases both factors are concerned and must be carefully investigated. There is an undoubted hereditary predisposition. Many infants in the same family, though well nursed and looked after, suffer. In some of these families there is an arthritic diathesis, a history of gout or allied states, and there is little doubt that this diathesis exercises some influence, possibly through circulating toxins. In others there is a history of neuroses, hay fever, asthma or rheumatism. The disease is rare in the tuberculous.



In many cases there is no evidence of dietetic error. Overfeeding and unsuitable food are apt to disturb normal metabolism, set up intestinal irritation and catarrh, and lead to the production of toxins or by-products of digestion. The disease is rare in marasmus and not infrequent in exceptionally fat babies. Possibly the exciting factor is some peculiarity in the milk, even in breast-milk, or the excess of sodium chloride in cows' milk. More often it is the excess of protein or fat in the milk, or some error in the maternal diet. These breast-fed sufferers are generally overfed, fat and anæmic. Combined with the overfeeding is deficient elimination. The bowels are constipated, the renal secretion insufficient, the liver overloaded, and the stools white and offensive. This state of the liver and stools is most common in infants fed on proprietary foods. In older children, the dietetic cause, if any, is usually an excess of milk and carbohydrates.

External causes are predisposing and exciting. Eczema due to external irritation is a *traumatic eczema* and unconnected with constitutional eczema. Atmospheric conditions of heat and cold, east and north-east winds, cause "chapping" of the face and hands, a "dry eczema." Hard water, strong and caustic soaps, powders, ointments, sweat, dirt, irritant discharges and unsuitable clothing are common causes. Fever increases the liability by the suffusion of the skin which it induces.

External irritants include antiseptics, arnica, etc., those of certain plants such as the *primula obconica*, of caterpillars, and of the various forms of trade dermatitis.

The *caterpillar rash* is set up by the hairs of the caterpillars and cocoons of various moths, notably the tiger (woolly bear caterpillar), silver-y, golden-y vapourer, gold-tail, silver-tail, brown-tail, drinker and oak-egger moths. The poison is either formic acid, or a substance allied to cantharidin, in the hairs. The rash is a dermatitis, eczema, impetigo or urticaria. It often begins with redness and itching, and develops into small papules, vesicles and pustules or into wheals. Apply an alkaline or lead lotion.

No definite microbial cause of eczema has been found. The vesicle is sterile at first. Unna's morococcus is a variety of *staphylococcus albus*. Microbial infection produces secondary pustular dermatitis. Vaccination is sometimes blamed, for the fever induced may stir up a seborrhœa into an acute eczema. Sometimes it has a beneficial effect.

Reflex causes are of little importance. Teething receives its mead of blame. Facial eczema may break out or increase in severity with the eruption of a tooth, but this does not prove reflex action. Worms are more apt to cause eczema through their toxins than by reflex irritation.

*Symptoms.*—In *dry eczema* or *chapping* the skin presents cracks and a moist exudation which crusts and irritates. On the cheeks it gives rise to marked redness, *E. rubrum*, and is chronic. This may spread to the forehead, ears and neck, scalp and trunk. The skin is red and swollen.



Small papules form, coalesce, and produce a moist exuding surface. Itching of the face and head is severe, and leads to scratching and bleeding, secondary infection and pustulation (*pustular eczema*). There is no such disease as a true pustular eczema. It is a pustular dermatitis from local irritants or infection, generally on the scalp, and like impetigo. Crusts form and on removal leave an inflamed granular surface, discharging serum and sero-pus, and bleeding readily. Or the eczematous process may not progress beyond the stage of redness, thickening of the skin, scaliness and itching. Restlessness and insomnia result from the itching and discomfort. Glands may enlarge and suppurate.

*Acute eczema* is more common in males than females, begins in the second to fourth month, is as frequent in the breast-fed, is often unassociated with gastro-intestinal irritation, and in 90 per cent. is a seborrhœic eczema spreading from the head. Occasionally it is secondary to dry, scaly eczema, to some parasitic affection, or of constitutional origin. The exciting cause is generally a trivial one, acting with undue virulence because of the predisposition in the skin or the constitutional state. The patient is almost invariably a fat baby with blue eyes and smooth, delicate skin.

The distribution in infants varies. In the first stage there is diffuse redness and roughness of the cheeks and forehead. It may be limited to these regions, to the malar bones, the upper lip, chin or behind the ears. The patches show minute papulo-vesicles, which becomes confluent and spread, with cracking and oozing, local swelling, heat and redness, and the formation of yellowish crusts. It next extends over the scalp, unless it has spread from there, and over the face, but avoids the skin of the nose, round the eyes and the mouth. Crusting is very marked. Patches appear on the forearms and calves, and sometimes practically all over the body. The general appearance and symptoms are the same as in adults, but there is greater tendency to pustulation. Large scabs form, suppuration extends underneath them, septic absorption ensues, and superficial ulcers are left when the scabs fall off or are removed.

The irritability may be so great that the infant is worn out by want of sleep. General health is lowered by septic absorption. Itching may be unbearable and paroxysmal. Glandular suppuration is not uncommon. Vomiting, diarrhœa, malnutrition, bronchitis, asthma and nephritis are the chief complications. Fever and convulsions occur, and occasionally sudden death, "*eczema death*," from cardiac failure or dyspnœa.

*Diagnosis*.—Exclude parasitic causes and external irritants. Examine for seborrhœa. A papular rash on the buttocks may be mistaken for congenital syphilis. Psoriasis must be differentiated.

*Prognosis*.—Recovery depends on the duration, the treatment and a careful nurse. It usually takes months, especially if there are digestive troubles. Acute infantile eczema is very liable to relapses. It rarely persists after the first year and practically never beyond the second year.



“Driving-in” the disease is harmless. Death may be unexpected, from status lymphaticus or from weakness of the cardiac muscles, the result of toxæmia and secondary infection.

*Treatment.*—Traumatic eczema is curable by local measures. In other varieties constitutional treatment is also necessary. Attend to the diet, the alimentary tract, and to eliminaton. If the child is breast-fed, diet the mother carefully on ordinary lines and let the child be nursed only 5 times in 24 hours, giving barley water beforehand, if it is desirable to reduce the diet. If bottle-fed on cows’ milk, reduce the percentage of protein. If this fails, reduce the fat. Milk can be omitted for 2-4 weeks, if the child is over 6 months old. If the child is overfed on carbohydrate foods, reduce or omit them for a time. Unsalted milk can be tried. Curdle 1 quart with rennet. Wash the curd several times with water and pass it through a sieve. Replace four-fifths of the whey by barley water and add dr. 6-8 of sugar. It makes a creamy soup. For older children reduce the amount of milk and carbohydrates; omit meat extracts, broths, tea and coffee, salt foods, and possibly eggs; and allow raw fruit and plenty of vegetables. In fact the general principle of diet is its limitation to a minimum quantity of the simplest foods.

Give alkaline waters, citrates and acetate of potash for the kidneys; rhubarb and soda if the stools are acid and fermented; or keep the bowels open with calomel, gr. 1-20 to 1-12 every 2 hours, or a dose of grey powder every 3-5 days. Vin. antimon. and mag. sulph. are useful at the onset of an acute case. Salol,  $\beta$ -naphthol and benzo-naphthol are the best intestinal antiseptics. Iron tonics and ol. mor. are needed in convalescence; arsenic in chronic cases.

The underwear must be of silk, fine linen or cotton, with wool over it. The child must be kept in bed in acute stages. Fresh air, without undue exposure, and sunshine are beneficial. The country is better than the seaside, except for secondary malnutrition and adenitis. Soft water is necessary.

*Local Treatment.*—Protect the parts from exposure, covering the face with a mask of lint, making holes in it for the nose, mouth and eyes. Allay inflammation and dry up secretions by muslin strips soaked in calamine lotion (p. 1001), weak alum lotion, or resorcin 1-2 per cent., and bandaged on. To relieve itching give a bath of bran, starch or gelatine and water; or a lotion of borax dr. 1-2, or creolin dr. 1, in water 2 pints; or calamine lotion, with carbolic acid 1-2 per cent. or ichthyol 0.5-1 per cent.; or apply lotio nigra freely on absorbent wool for 10-15 minutes several times daily. Powder with zn. stearate. Cleanse with carbolised oil 1 per cent. Warm soft water must be used for washing and an animal fat rubbed in after drying. Neither soap nor water is permissible in the acute vesicular stage, for it removes the protective exudation. The morning bath can be given in mild cases, if the affected parts are protected by ointment.



To prevent scratching cut the nails short, put on gloves, cover the parts with dressings and bandages, and use restraining splints. And give internally antimony at the onset, bicarbonates, calomel, chloral, ichthyol, phenazone, gelsemium or hypnotics.

Crusts are removed by soaking them with warm olive oil for 12-24 hours, normal saline, weak bicarbonate solution, carbolised liquid vaseline, boroglyceride fomentations, dr. 1 ad 1 pint, bread or borated starch poultice. It is no use applying ointments during the oozing stage or on the top of crusts.

Infection is prevented by cleanliness, protection from exposure, the removal of scabs, the prevention of scratching, and the use of creolin lotion (dr.  $\frac{1}{2}$ -1 ad 1 pint) or salicylic acid ointment. In extensive cases clean with weak warm gruel, boroglyceride lotion or a creolin bath. Subsequently cleanse with warm carbolised oil 1 per cent. and pledgets of wool. A tar bath can be used for extensive pustulation or impetigo; starch poultices and creolin lotion for less severe cases.

To dry up secretions apply dusting powders (p. 999). They are easier to remove, and useful for keeping opposed surfaces apart, when placed in flat muslin bags. They must not be dusted on the scalp because of the difficulty in removal.

For protection and stimulation of the surface apply on lint, fixed on with a soft bandage, a cream or paste (p. 1000) containing zn. oxide; or the zinc cream recommended by Malcolm Morris, consisting of lanoline dr. 1-2, ol. olivæ oz. 1, aq. calcis oz. 1, zn. oxid. dr. 6-7, a thick cream in which strips of muslin or old linen are soaked. Ichthyol dr.  $\frac{1}{2}$  can be added, if desired. Or use Lassar's paste with ac. salicyl. gr. 10-20, sulph. præcip. gr. 10-30, or with calomel, bismuth, subnit. or resorcin gr. 10-20 ad oz. 2.

In the dry scaling stage wash with creolin lotion and apply liq. carb. detergens or alcoholic solution of coal tar dr.  $\frac{1}{2}$ , hydrarg. ammon. gr. 10, vaseline oz. 1-2; or zn. oxid. 1, ung. picis liq. 2, ung. aq. rosæ 4 parts. Ointments of tar, sulphur, salicylic acid, resorcin or mercury are now suitable. Coal tar preparations must be used with care, and strong antiseptics avoided, in infancy. If it is desirable to omit bandages, use a zinc gelatin or plaster mull; or Pick's paste (p. 1000) with zn. oxide gr. 40, carbolic acid gr. 5, or tar m. 10 per ounce. For pustular dermatitis ung. hyd. amm. 1, lanoline 1, vaseline 3-6 parts is a suitable application.

**Psoriasis.**—A family history of the disease is often present and has been traced through 3 generations. The position in life is unimportant, the general health excellent, and the sex-incidence about equal. Possibly a gouty or rheumatic heredity has some influence. Attacks sometimes follow vaccination and exanthemata. The age-incidence is roughly 50 per cent. under 15 years, 20 per cent. under 7 years, and 5 per cent. under



3 years of age. A few cases begin in the first year, e.g. at 5 and 38 days (Rille), 3 months (Billard), 4 months (Newmann). It is most common in spring, autumn, winter and cold weather. Possibly it is influenced by lack of sun and light. Attacks may occur annually or at frequent intervals.

It often begins with one or two isolated patches, spreads locally, and finally becomes general through blood infection or increasing metabolic changes in the system.

According to the size of the patches it is named *P. punctata*, *guttata*, *nummulata* or *diffusa*. These are merely stages in its development. During involution the spots clear up at the centre and leave a circinate or gyrate outline. In children the patches are small, punctate, guttate or nummular, round or oval, showing abrupt transition to healthy skin. The scales may adhere into a crust, and on removal leave a red base. The rash is widely distributed, not limited to the extensor surfaces, and may even involve the penis. The face and palmar surfaces of the hands and feet usually escape. On the whole the extensor surfaces and dorsal region are most involved, and the rash diminishes peripherally. The scales are small and easily detached, varying in colour with that of the skin. On the scalp it is difficult to distinguish from seborrhœa sicca, but generally an isolated patch can be found which is covered with thick white, silvery, not greasy, scales. Bleeding spots are left on scraping off the scales. The *nails* are brittle and cracked; not thickened, but tilted up by accumulated epidermis, due to chronic inflammation of the nail bed.

*Treatment.*—Cure every patch, not merely the bulk of them, for recrudescence is common. Internal remedies seem of more value than local ones for permanent benefit. The best drugs are rhubarb and soda, alkalies and diuretics, nitric acid after food, salicin and tonics; and arsenic in chronic stages. A cooling lotion or ointment, as in eczema, may be needed in acute stages.

Give an alkaline bath of sod. carb., of creolin dr. 1 in 6 gallons, or a warm bath for 10-15 minutes with sulphur or soft soap; remove scales by scrubbing; and apply to each spot liq. carbonis detergens, ung. ac. salicyl., or an ointment of ac. salicyl. gr. 10-20, hyd. ammon. gr. 10, creolin dr. 1, vaseline oz. 1, to which may be added chrysarobin gr. 10, sulph. præcip. gr. 10, soft soap dr. 1-2. Ung. picis liq. 1, vaseline 7 parts, is useful, smelly and dirty. Ol. cadeini dr.  $\frac{1}{2}$ -2, vaseline ad oz. 1, is preferable. Chrysarobin is most efficacious, but stains everything and may cause severe erythema or acute conjunctivitis. It makes the patches white and the sound skin red. White precipitate ointment is then rubbed in. Chrysarobin gr. 5-10, ac. salicyl. gr. 10 in traumaticin oz. 1, can be painted on each spot. It forms a film which does not stain when dry. Resorcin, naphthol, and other tar preparations are also of value. For the head use sp. saponis alk., rubbed in at night and washed out in the morning; or hyd. amm. gr. 10, soft soap and vaseline p.a. oz. 1, rubbed in nightly after washing;



or ac. salicyl. 5 per cent. in ol. ricini and ol. olivæ p.a., to remove the scales.

The sulphur baths of Harrogate, Strathpeffer, Aix-la-Chapelle and Barège in the Pyrenees, and the arsenical waters of La Bourboule, Levico and Roncegno in the Tyrol are more suitable for chronic cases in adults.

Most cases in children are curable in 4-6 weeks. Dietetic treatment is sometimes needed. Bulkley (1908) found the urine abnormally acid and containing a double quantity of urea. He states that meat diet accentuates the rash, and prescribes a true vegetarian diet, excluding milk, eggs and fish. Such a diet is unsuitable and unnecessary in childhood.

**Scleroderma** is localised or generalised. In the local type there are patches of dull or glossy, ivory white, hard skin; often stiff, thickened and painful; sometimes symmetrical, though no two patches are exactly alike, and called *herpetiform morphæa*. It disappears in time, leaving the skin a little thin, brown and shrivelled. In a more severe type the cellular tissues, muscle and bones are affected and arrested in their growth, e.g. facial hemi-atrophy. The generalised variety is a type of *Sclerema*. The skin is thick, dry, harsh, immobile and parchment-like, without leucoderma or pigmentation. Its natural folds are obliterated, the facial expression is lost, and there is a more or less widespread, "hide bound" appearance. It may be associated with inflammation, atrophy and contraction of muscles. Possibly it is an infection of the subcutaneous tissues and muscles, ending in sclerosis. Its course is progressive. Some patches may recover and others appear. Persistent massage with emollient ointments is beneficial.

**Corns** are more troublesome in adults than children, except in the form of corneous thickenings of the heels and soles from unsuitable boots. For these apply salicylic acid gr. 30, ext. cann. ind. gr. 5, collodion oz. 1, nightly, and pare off the thickened skin. A lotion of soft soap oz. 2, methylated spirit oz. 3, applied on lint, will prevent the corn re-forming.

**Warts** are often multiple, cause little or no discomfort, and may disappear spontaneously. They are most numerous on the hands, fingers and scalp. Apparently they can be transmitted, and have an incubation period of 1-8 months. Disappearance has followed the taking of lime water, oz. 1-4, t.d.s. Local applications include wearing gloves dusted with calomel and French chalk; saturated caustic soda solution daily; ac. salicyl. 1, ac. lactici 1, collodion 2 parts, bis die; papain dr. 1, ac. hydrochl. fort. dr. 2, bis die; glacial acetic acid; chloral hydrat. gr. 15, ac. acetic. m. 15, ac. salicyl. dr. 1, ether dr. 1, collodion dr. 4, daily; sulphur sublim. 2, glycerine 5, acetic acid 1 part, painted on several times daily; ol. terebinth. half-hourly and dried in before the fire; the galvanocautery, electrolysis, X-rays, re-vaccination and suggestion. After a single application of X-rays for 15-30 minutes, the wart drops off in 7-10 days, leaving smooth healthy skin, with no scarring.



**Molluscum Contagiosum** is due to the metamorphosis of the cells of the rete Malpighii into keratin. Probably it is started by a parasite which sets up an in-growing epithelial proliferation of the duct of a sebaceous gland. It may become inflamed. Small, waxy, umbilicated tumours, like little buttons, are found in variable numbers on the forehead, face, round the eyelids, and sometimes on the neck, limbs, genitals and round the anus. A peculiar core can be expressed, on making a small incision. The treatment consists of curetting after expression of the core. Or touch the apex of each tumour with a drop of pure carbolic acid. After a few applications it withers and falls off.

**Lichen Planus** is a rash of pinkish, flat-topped, shiny papules, which may be aggregated into pavement-like plaques. It may affect the mucous membranes. It especially appears on the extensor surfaces of the arms and thighs. The palms and soles are usually free. Arsenic or biniodide of mercury should be given; and antimony, if arsenic exaggerates the symptoms. *Lichen spinulosus* consists of groups of spiny papules.

**Alopecia**.—General thinning of the hair after illness is treated by tonics and rubbing in chloral hydrat. gr. 10, ung. hydrarg. ox. rub. dr. 1-2, lanolin ad oz. 1. True alopecia is characterised by partial or complete loss of hair, due to a neurotrophic or toxic affection of the hair papilla. When universal it begins in patches, which are asymmetrical and immune from inflammatory reaction. No parasitic cause has been found. The contagious cases are probably varieties of ringworm. The bald patches are pale, bloodless, smooth, shining and circumscribed. Short hair stumps at the edges are thick and pigmented at the free end, thin and pale at the attached end, like “notes of exclamation.” The hair root is atrophied, but the hair is more adherent than in ringworm. The course is very slow. The prognosis is better than in adults. The longer the growth of new hair is delayed, the worse is the prognosis. Fine downy hairs grow and become gradually stronger and darker. Relapses are not uncommon.

Tonics and intestinal antiseptics may do good. The main treatment is local irritation by massage, faradism and drugs, e.g. ol. cinnamon. 1, ol. olivæ 4 parts; ol. sinap. dr. 1, ol. ric. dr. 2, sp. rosmarini ad oz. 4; liq. amm. fort., chloroform, ol. sesam. aa. dr. 2-4, ol. limon. dr.  $\frac{1}{2}$ , sp. rosmarini ad oz. 4; bals. Peru, the tinctures of capsicum, iodine, veratria and cantharides, pure carbolic acid, ung. chrysarobin 0·5-10 per cent., ung. hyd. ox. rub., etc., in sufficient strength to produce mild inflammatory reaction.

**Sudamina**.—The rash consists of multiple, minute, transparent vesicles, or minute pustules, due to the accumulation of sweat in glands whose ducts are blocked by dirt or epithelial cells. It is the result of profuse sweating. There may be a general erythematous base or intervening minute red papules. On the palms, soles and sides of the fingers it appears as minute, whitish, itching papulo-vesicles. The rash often itches and is



followed by slight desquamation. Apply vinegar and water, dusting powder, and the measures used for intertrigo.

**Strophulus**, *red gum* or *miliaria rubra*, is an eruption of scattered red papules, due to inflammation of sweat glands by retained secretion. The name, strophulus, is occasionally given to inflamed sudamina and to lichen urticatus. Sometimes tiny vesicles are formed and the rash is analogous to prickly heat. Probably there are two kinds of "red gum," one due to inflammation of the sweat glands and the other to disturbance of function of the sebaceous glands. Frequently there is a good deal of erythema and the spots resemble insect bites. They are distributed in irregular groups, appear on the face, are often abundant on the front of the forearms, just above the wrist, and are absent from the hands. They disappear spontaneously. Some of them are hard, with a "shotty" centre, like abortive pustules. The rash bears no relation to the state of health of the child, the diet or the clothing. No special treatment is needed.

**Acne**.—*Comedones* or "black-heads" are not due to mere plugging of the ducts of sebaceous glands with dirt and sebum. Each one consists of concentric layers of horny cells, enclosing a mass of the micro-bacilli of acne, which find in the oily sebum a suitable soil. This horny plug sets up irritation and, as the result of staphylococcal infection, gives rise to the papules or papulo-pustules of acne. Hence acne consists of three stages, viz. greasy skin, comedones, and folliculitis from secondary infection.

Grouped comedones are common in infants and children, especially on the forehead, cheeks and chest. Sometimes they are due to the application of camphorated oil, Russian tallow, etc., and consequent blockage of the ducts.

*Acne* is common about puberty, rare in younger children. Single spots can be touched with liquefied carbolic acid, or incised and washed out with carbolic acid 20 per cent. For multiple acne bathe well with hot water and apply a good lather of ichthyol 10 per cent. soap, night and morning, during the inflammatory stage. Then rub in nightly an ointment of sulphur præcip. gr. 15, hyd. amm. gr. 5, hyd. sulphid. rub. gr. 5, vaseline ad oz. 1.

Prevention consists in keeping the skin free from excessive sebum, squeezing out comedones unless inflamed, and rubbing in lysoform soap lather nightly with a shaving brush. Lotions are often preferable to ointments, e.g. sulphur. præcip., glycerin., ether aa dr. 2, sp. vin. rect. dr. 4, aq. rosæ ad oz. 10, mopped on freely at night and allowed to dry on. Give a morning saline aperient, regulate the diet, and prohibit alcohol. Internally give the sulphates of Fe. and Mg., and tonics; and yeast, ceridin, or calcium sulphide if there is suppuration. Disinfect the skin, and try vaccine in severe cases.

**Furunculosis**.—A boil runs the same course as in adults. Small ones, containing yellow or greenish-yellow pus, appear in great numbers



on the scalp, buttocks, thighs and shoulders of marasmic infants, sometimes in successive crops for months, and occasionally in the well-nourished. They are due to infection of the skin by the staphylococcus aureus, and may become gangrenous or set up pyæmia. Sulphur baths, ointments and pastes are the best remedies.

Prescribe yeast, the ferment of beer or grapes; calcium sulphide, hypophosphites, ferri-ichthyol, arsenic, iron, ol. mor., or quinine. Feed well and attend to the digestive tract. Inject locally, to induce abortion, tr. iodi or pure carbolic acid, m. 1-3. To relieve pain and encourage suppuration, apply boric acid fomentations or starch poultices. Rub an antiseptic ointment into the unaffected skin. Spontaneous rupture leaves the least scar. In unimportant places incise and apply antiseptics. A satisfactory method of treatment is the application of glyc.ac.carbol. on wool, covered by guttapercha tissue. As the boil points the epithelium is reflected and a little of the glycerine injected. The slough separates in 2-3 days. Or paint the boil, except the apex and the surrounding skin, with collodion, flexile 2 and non-flexile 3 parts, covering the whole with absorbent antiseptic wool. As the collodion contracts it squeezes out the core. A preliminary small incision may be required.

**Impetigo Contagiosa.**—Pustular dermatitis may occur in the newborn and cause sepsis. Impetigo is a local form, usually due to staphylococcal infection. It chiefly affects the face, head and hands, and is spread by scratching. It is contagious. Staphylococci have been grown in pure culture from unopened bullæ and have experimentally produced the disease; so, too, streptococci. The rash begins with erythematous spots, which become vesicles. These soon contain sero-pus or pus, and have a slightly red areola. The pustules rupture and discharge, and thick yellowish scabs are formed, leaving a moist red surface on removal. The sores may become circinate through healing centrally and spreading peripherally. Wash with a weak antiseptic lotion. Open vesicles and clean the skin. Remove crusts after softening them. Apply sulphur or white precipitate ointment, or a sulphur lotion. Give ol. mor. and iron.

**Ecthyma** is closely allied to impetigo, but the base is infiltrated and leaves a pigmented scar. It is most common on the nates and lower limbs, rare on the trunk, and is secondary to scratch infection. In the gangrenous form, like gangrenous varicella, deeply cut ulcers are formed, with a black slough at the base. The treatment is the same as in impetigo.

**Pemphigus.**—This is a bullous eruption, occurring sporadically or epidemically, and like pemphigus neonatorum (p. 142) in its pathology. It comes out suddenly and rapidly all over the body and limbs, but is rare on the palms and soles. It begins on the extremities, trunk, buttocks, or perhaps on the eyelids. Though somewhat allied to herpes the rash has no definite nerve distribution. At first the bullæ are like those of simple scalds. Later they show a narrow red areola, due to inflammation. They vary in



size from a pin head to a crown piece. Usually they are not very numerous, sometimes only a few, but occasionally almost the whole body is covered and they may coalesce. Their contents are clear or cloudy serum, which may become purulent. They are absorbed, or the bullæ burst in 1-3 days and leave a raw surface which soon becomes thinly scabbed. After the scab falls off some discolouration is left. The rash may affect the mucous membrane, causing swelling of the gums and intense congestion of the buccal mucosa. Bullæ in the small and large intestine give rise to abdominal pain and diarrhœa. Eosinophilia is generally present.

In mild cases there are no symptoms. In severe ones, sometimes fatal, there is high fever, restlessness, anorexia, vomiting, rigor or chilliness, offensive urine and albuminuria. Even apyrexial cases may prove fatal. The rash lasts from 2-7 or more weeks; may appear in crops; may be recurrent, or chronic with exacerbations; and may be followed by complete desquamation, leaving the cuticle red and weeping. Death is rare.

Protect the parts from injury and sepsis, by means of antiseptic dusting powders and lotions. Prick the vesicles. Relieve pain. Give bran and oak bark baths. Arsenic is the most useful drug.

**Herpes.**—*Syn.* : *Herpetic fever*—*Herpes zoster*—*Shingles*—*Labial herpes*—*Zona ophthalmicus*.—This is a general disease with a local manifestation rather than a simple skin affection. It is divisible into a :—(1) Specific or infective variety, an acute specific infectious disease; (2) Toxæmic type, of digestive or febrile origin; (3) Secondary variety. The most probable explanation is that the rash is due to neuritis and that the neuritis depends on many causes. Barendsprung (1861) found a lesion in the posterior root ganglion, and ascribed it to irritation of the spinal or Gasserian ganglia, and occasionally to peripheral nerve irritation. Head and Campbell (1900) discovered an acute inflammatory condition of the ganglia in acute cases, and secondary degenerative changes, not demonstrable before the eleventh day, in the posterior nerve roots and peripheral nerves. Changes take place in the spinal cord, similar to those following section of the posterior root. The area of the rash corresponds with that of the affected posterior root ganglion. Complete recovery, or sclerosis and persistent pain, may ensue.

*Primary herpes* is the result of an infection of the posterior root ganglia which irritates the nerves and causes the rash. *Secondary herpes* is due to these ganglia being implicated by injury, disease, or drugs such as arsenic.

The specific variety is in many respects analogous to acute poliomyelitis, except that the poison attacks the sensory and not the motor parts of the nervous system. There is a distinct prodromal period. The rash comes out on the third or fourth day, sometimes within a few hours, and is not always localised. It occurs in epidemics, chiefly in spring and autumn. In true herpes zoster second attacks are infrequent. Relapses occur in



about 1 per cent. On the other hand there are cases which are markedly recurrent and probably non-specific. Thus, in 4 girls I have known frequent recurrence on the lips or cheek. Ophthalmic zona has recurred 3-4 times a year from childhood (Matignon).

*Symptoms of Shingles.*—After a variable period of malaise, digestive disturbance, chilliness or rigors, and fever, a pain develops along certain nerve paths or metameric areas. It is often severe, burning, itching, stabbing or neuralgic, and may be ascribed to pleurisy or pneumonia. The lymph nodes are enlarged, sometimes before the eruption. In 1-5 days the rash comes out along the course of a cutaneous nerve. At first it is papular, but the papules quickly develop into clear vesicles, of the size of a pin head to a split pea. Each vesicle is surrounded by a red areola and these areolæ may coalesce. The rash is more or less continuous, large groups of vesicles being linked together by smaller ones or single vesicles. Their contents are sterile, become sero-purulent, and soon dry up, leaving scabs in 1-2 days. The pain usually disappears with the eruption of the rash. Fever subsides quickly and recovery is rapid. Fever may persist for 6 days. The blood shows leucopenia, a reduction of polymorphs to an average of 45 per cent.; and moderate eosinophilia, 6-8 per cent. The complications are slight adenitis, extensive ulceration and gangrenous dermatitis.

Labial herpes is common in digestive disturbance, pneumonia and cerebrospinal fever. In children I have seen herpetic eruptions on practically every part of the body, except perhaps the palms and soles. They are less frequent on the shoulders and limbs than in adults. Occasionally it attacks the mucous membrane of the mouth, pharynx, larynx, nose or genitals. It is rare under 4 years of age. The sex-incidence is twice as great in females. Shingles is rarely bilateral and is limited by the middle line of the body, but not rigidly, for the nerve filaments sometimes encroach over the border. Fever and gastric symptoms are the common signs.

French dermatologists have described “isolated vesicles disseminated over the body in herpes zoster.” Aberrant vesicles of this type have been noted by English observers. They are about the size of a hemp seed, with a red areola, like varicella.

*Treatment.*—Keep the child in bed until the fever has subsided. Give a calomel purge, mild febrifuge and light diet. Paint the rash with collodion, or dust on equal parts of calomel, zinc oxide and starch, and put on a pad of absorbent wool to protect it from injury and prevent rupture of the vesicles. Treat secondary ulceration and gangrene on ordinary surgical principles. Give hypophosphites, arsenic and cod-liver oil in convalescence.

**Tuberculosis of the Skin.**—The bacillus has been found in lupus vulgaris, lichen scrofulosorum, scrofuloderma or tuberculous gummata, and acute miliary tuberculosis of the skin, a rare eruption of acneiform papules. Lupus vulgaris may occur after measles in a nodular form on the



face, trunk and limbs. *Lichen scrofulosorum* consists of an insignificant eruption of very small, pink, follicular papules in circinate patches on the abdomen and limbs. It lasts for months, but may finally disappear and leave pigmented scars. *Tuberculous gummata* are small deep-seated abscesses, which sometimes burst spontaneously, and are an advanced stage of *Acne Scrofulosorum*. This consists of one or more papules, up to a pea in size, at first subcutaneous, and later dusky red or purplish and involving the skin. They often break down, forming small punched out ulcers which heal and leave a circular scar. They are generally situated on the limbs, run an indolent course, and may slowly disappear. The bacillus is said not to have been discovered in these nodules. Scrapings from one, taken from a well-nourished boy, produced tuberculosis in guinea pigs. Many of these children are fat and appear healthy. One such child under my care died from tuberculous meningitis, and caseous nodules were present in the brain.

*Erythema Induratum vel Scrofulosorum* (Bazin's disease) is apparently a later stage of the last variety. Deep-seated, painless, dusky or bluish plaques and nodules are found in the legs, generally the calves, of delicate girls who stand a great deal. They are apt to ulcerate. Scrapings have produced inoculation-tuberculosis in guinea pigs. The treatment is essentially that of tuberculosis and that appropriate for the nodules and ulcers, if any. Gummatus broken down nodules should be curetted and swabbed with pure carbolic acid.

## PARASITIC SKIN AFFECTIONS.

*Ringworm—Tinea Tonsurans—Favus—Insect Bites—Harvest Bugs—Pediculosis—Scabies—Creeping Larvæ.*

**Tinea**, or *ringworm*, has been known for centuries. Gruby, of Paris (1843), first ascribed *tinea tonsurans* to a cryptogamic fungus which he named the microsporon Audouini, after Audouin who had recently described such a fungus in silkworm disease. Malmstein, of Stockholm (1844), discovered a similar fungus and called it the trichophyton tonsurans. Sabouraud has made extensive and valuable researches, and has described (1908) 11 varieties of fungus. By far the most common is the microsporon. It is present in 60-65 per cent. of the scalp cases in Paris, and in 90-95 per cent. of those in London and Edinburgh. The remainder are due to a large spored fungus in the form of threads, *trichophyton megalosporon endothrix* and *ectothrix*. The *ectothrix* variety, in which the fungus lies outside the hair, between the dermic portion of the shaft and the hair follicle, is the most curable. The species conveyed from the horse is very



liable to set up suppurative lesions. Ringworm of the body is almost always due to a trichophyton.

*Etiology.*—The botanical character and origin of the fungus are uncertain. Probably trichophytons exist saprophytically. The ectothrix is generally communicated by domestic birds or animals, and the microsporon from another child, horse, cat or dog. Ringworm is transmitted by direct contagion or through the medium of caps, brushes, etc. Both sexes are equally liable. Body ringworm may occur at any age. Scalp ringworm, due to the microsporon, is peculiar to children and does not persist in adults. Fair-haired are more susceptible than dark-haired children.

*Pathology.*—In the common type the fungus grows first in the epithelium, forming small greyish scaly patches. It gradually invests the hair with a white or greyish mosaic sheath of spores, while the mycelia invade its substance. The hairs extend 2-3 mm. beyond the orifices of the follicles, and are finer and longer than in the trichophyton infections. In the endothrix variety they are broken off short, level with the scalp, “black-spot” ringworm, and show no trace of a sheath. The fungus is within the hair, the spores are arranged in chains, and the patch is free from scales. In the ectothrix type the hair and epidermis are affected, and the dermic portion of the hair has a sheath. The fungus is really an endo-ectothrix for it invades the shaft, erodes it from within, and causes the hair to break off close to the mouth of the follicle. The microsporon erodes the shaft from outside and the hair breaks off a short distance from the surface. In cultures on agar maltose 6 out of the 11 varieties parasitic on man and animals (Sabouraud) are small-spored, like the microsporon, and form large white powdery growths; 4 are large-spored and form large white downy cultures; and the microsporon audouini forms disc-shaped, downy or woolly growths.

*Mode of Examination.*—Extract a hair from the edge of the patch by gentle traction in its long axis, so as to get as much as possible of the bulb. Put it in warm liq. potassæ for  $\frac{1}{2}$  minute, transfer it to fresh water and then to glycerine, and examine under a  $\frac{1}{4}$ - $\frac{1}{6}$  in. objective. For staining, soak it in ether for 5 minutes to dissolve out the fat; stain in a dye of gentian violet 5 per cent. in alcohol, 70 vol. strength, for 5-60 minutes, with gentle heat; decolourise with Gram-iodine solution, clear with anilin oil, wash in xylol and mount in xylol balsam. The spores are stained blue. For red staining use carbol-fuchsin, 2 per cent. The microsporon stains the most readily.

**Tinea Tonsurans** is extremely prevalent, almost peculiar to children, and entails exclusion from school or education in special schools. It is most common at 5-10 years of age, may occur in the first year of life, decreases in frequency after the tenth year, and virtually ceases at puberty. Boys are more often infected than girls, through interchange of caps. The incubation period is about a fortnight.



It starts round a hair follicle as a small papule which spreads and forms a round or oval, slightly raised, scaly patch, dirty grey or yellowish to reddish brown in colour. A microsporon patch is round, sharply defined, and has a narrow red areola. It is studded with short, broken, atrophied hairs of a dirty white appearance, because of their mycelial sheaths. They project as cone-like knobs, causing a goose-skin aspect of the patch. The hairs are loose and can be pulled out without pain, and are thickened from infiltration with fungus.

The short stumpy hairs have been described as like "bent fingers" or "stubble in a cornfield." They are irregular in growth and direction, and taper off at the free ends or finish off abruptly. In the megalosporon type the stumps of the hairs are broken off at a level with the skin and look like black dots.

Sometimes the fungus is disseminated in numerous isolated patches all over the scalp, or there may be a general scaliness liable to be mistaken for pityriasis or seborrhœa. In another variety, *tinea decalvans*, the hairs fall out and leave smooth patches like those of alopecia, but broken hairs are found at the edges and many of the follicles are indicated by black dots. An impetiginous form exhibits scabs and crusts, through which broken hairs often project. In *kerion* the patch is inflamed, raised, boggy and fluctuating, and exudes pus. It is the result of too active treatment, inoculation with pus cocci, a specially virulent ectothrix infection, or an attempt at cure by nature. The follicles are filled with broken hairs or pus, and occasionally a subcutaneous abscess forms, but there is no sloughing. Permanent baldness may ensue. Kerion, impetigo and inflammatory patches like boils are the only complications.

*Course.*—In the young it spreads rapidly and may involve the whole scalp in a week or two. The disseminated type is persistent, because small patches are apt to escape cure. In chronic cases the orifices of the follicles are blocked and it is difficult to reach the enclosed fungus. Baldness is rare, except as the result of treatment or kerion. The latter works its own cure, for the purulent inflammation destroys the fungus. The older the child, the more quickly is it curable. Untreated cases recover after puberty. The microsporon variety is the most intractable and may persist for 3-6 months. *Tinea decalvans* is curable in a few weeks. Chronic thickening and the effects of injudicious treatment prolong every case.

**Tinea Circinata.**—This occurs at any age and in either sex. It is most common in young children. It is due to the trichophyton, occasionally the microsporon. The fungus grows centrifugally, the mycelial threads spreading and causing erythema which fades at the centre as it extends peripherally, forming the characteristic ring-like patches on the skin. As the erythema fades the patches become scaly. After remaining stationary for a time they gradually die away. Sometimes there are patches but no rings; occasionally concentric or incomplete rings. Itching



may be present and lead to scratching and pyogenic infection. The rash is most common on the neck and face, hands and forearms, and the trunk. It tends to recovery but may persist for months, if untreated.

*Onychomycosis or ringworm of the nails.*—The fungus, generally an ectothrix, invades the nail and sets up inflammation. The nail becomes thickened, dull, uneven and brittle. Exfoliation occurs and under the free border is seen a mass of disintegrated nail structure. Microscopical examination is necessary to distinguish it from favus and psoriasis.

*Ringworm of the palms and soles* begins with vesicles and blisters which rupture and leave a bare pink surface. This becomes dry and scaly, spreads at the edges, and has a thickened border. The fungus is found in the fluid and scales. There is much itching.

**Treatment.**—Attend to the general health for it is often poor. Note the age of the patient, coarseness of the hair, and the extent, distribution and clinical characters of the disease. It is difficult or impossible to destroy the parasite *in situ*. Consequently epilation or depilation is desirable. Epilation is only suitable for small patches, and is even then unsatisfactory for many hairs break off.

Depilation is best carried out by means of *X-rays*. This method of treatment is undoubtedly the best, provided it is carried out by an expert. There is no danger of injury to the brain. In skilled hands the risks of dermatitis and permanent baldness are very small. The treatment must not be applied if the scalp is inflamed, especially if inflamed by iodine. Subsequent irritation, before the hairs drop out, leads to scratching of the scalp, pus infection, impetigo and folliculitis. The rays are not parasitocidal, but the fungus falls out with the hairs.

The hair is cut short, and the affected areas marked out with a blue pencil. Unaffected areas are protected by lead sheeting. Or the rays are concentrated by means of lead-glass cylinders, of suitable size, on the affected patch at a distance of 15 cm. from the source of the rays. The dose is measured by means of colour changes in pastilles devised by Sabouraud, placed 8 cm. from the source of the rays. It is better to give one full exposure than several milder ones for the risk of over-dosage is less, seeing that the effects cannot be measured for some time. The hair falls out in 14-21 days, grows again in 6-8 weeks, and is fully grown in another 3 months. Apply a weak white precipitate ointment to prevent infection by the falling hairs. Usually there is a little erythema a week after the exposure. The prolonged period of partial or complete baldness is a disadvantage. For extensive cases it is necessary to depilate the whole head, dividing it into 4-5 areas for separate exposure. Depilated patches become quite smooth. Diseased stumps, which have escaped, in the margins of patches may be treated by *croton-oil needling*. A needle coated with a film of the oil is gently inserted into the follicle, taking care not to pierce the skin. The hair can then be pulled out or is shed from pustular folliculitis.



Depilation by inflammatory treatment results in kerion. It causes discomfort, often pain; is unsightly and even alarming; and is difficult to regulate in extent. Croton oil, Coster's paste (iodine dr. 2, ol. cadeini oz. 1), liq. epispasticus, pure carbolic acid, chrysarobin, formalin, turpentine, hyd. iod. rub., etc., are used.

The croton oil method is the best, and is suitable for small patches. Cut the hair short for  $\frac{1}{2}$  in. round and rub carbolised vaseline into this area. Rub one drop of croton oil into the patch with a camel's hair brush and put on a linseed poultice in 3 hours, covered with oiled silk. Remove the poultice next morning, wash with warm water, prick the yellow blisters, and paint and poultice as before. Repeat this daily until kerion is set up. Then apply boroglyceride lotion to reduce the inflammation, and extract all the stumps. A red bald patch is left and downy hair grows in 6-8 weeks. Or an ointment of croton oil dr. 1, lanoline ad oz. 1, can be rubbed in daily, until the requisite amount of inflammation is set up and all the hairs have been epilated. Allow no scabbing.

A single application of blistering fluid cures some cases. In chronic ones it must be applied weekly, diluted with glycerine if blistering is to be avoided.

*A simple method.*—Cut the hair quite short for  $\frac{1}{2}$  in. round a patch or shave the whole head every 2 weeks. Rub in turpentine and then wash with soft soap or sp. saponis alk. to remove all sebum and epithelial debris. Repeat the soft soap washing every 2 or 3 nights. Mark out affected areas, and rub carbolised lanoline or glycerine (1 in 8) into the rest of the scalp. Keep on a linen head covering by day and night. A lotion of ac. salicyl. gr. 5-10 in ether or chloroform dissolves out fat, dehydrates the tissues, loosens hairs and attacks the fungus. It must be brushed in nightly.

Mild measures, such as just described, may cure recent cases and those due to the megalosporon. Other remedies of this type are a saturated solution of Na. Cl., or vaseline and Na. Cl. pa.; sod. hyposulphit. dr. 1-2, vaseline oz. 2; and saturated solution of boric acid in methylated spirit or ether, applied for 15 minutes daily.

More severe remedies include:—(1) Hyd. perchlor. gr. 10, acetic acid oz. 2, glacial acetic oz. 1, applied daily for 2-3 days; (2) Carbolic acid 1, balsam of Peru 1, petroleum 10, glycerine 10 parts, every 1-3 days; (3) Pure carbolic and iodine,  $\overline{aa}$  dr. 1, collodion oz. 2, painted on once or weekly; (4) Ac. carbol. dr. 1, ac. salicyl. dr. 1, vaseline oz. 1, rubbed in m. et n.; (5) Chrysarobin gr. 10-60 ad adip. benzoat. oz. 1, alone or with ac. salicyl. gr. 10-20, or hydrarg. amm. gr. 10-20, and a little ichthyol to reduce the inflammation; (6) Copper oleate gr. 30 in ung. hydarg. oleatis, 20 per cent., oz. 1; (7) Formalin, 10 per cent. solution, applied on 2-3 successive days, and followed by boric fomentations. It is often painful and may cause useless eczematous inflammation or much oedema. (8) Decolourised iodine,



made by adding calomel to the tincture and decanting the clear fluid, applied by camel's hair brush; (9) Mercurial ointments, unsuitable for very young children or those with delicate skins; (10) Sulphur præcip. dr. 1, ac. carbol. gr. 10-30, ol. olivæ and lanoline āā oz.  $\frac{1}{2}$ ; sulph. præcip. dr. 1, hyd. amm. gr. 20-30, ac. salicyl. gr. 5-15, ol. olivæ and lanoline ad oz. 1; (11) Naphthalin 20 per cent. solution; (12) Liq. ammon. 5, ess. terebinth, 25, camphorated alcohol 125 parts (Hallopeau), rubbed in daily after washing with soft soap, and followed by inunction of iodated vaseline, 1 per cent.

Successful treatment depends more on the care and thoroughness with which it is carried out than the actual parasiticide used. Drugs do not penetrate sufficiently deeply to kill the fungus, so epilation or depilation is essential, followed by the use of ointments which prevent superficial spread.

Ringworm of the skin is easily cured by painting with tr. iodi, alone or with the addition of 30 per cent. acetic acid or 5 parts of alcohol; or with Coster's paste. An ointment of hydrarg. amm., gr. 3 ad oz. 1, is suitable for young children. The *signs of cure* are the absence of broken hairs and of fungus on microscopical examination. Make a weekly examination for a month.

**Favus** is almost invariably due to the achorion Schonleinii. It is sometimes transmitted from mice, cats and dogs. Fortunately it is rare in this country, and chiefly seen among aliens in the East end of London. It begins round the hair follicle as a yellow deposit or plaque, which gradually increases in size until it is convex. The colour slowly changes to grey or dust-like. The odour is that of mice. The hair atrophies and drops out. Favus is chiefly limited to the head but may affect the nails. It is treated by X-rays, epilation, daily washing with a disinfectant, and the application of a mercurial or pyrogallic ointment.

**Insect Bites.**—Midges, gnats, mosquitoes and other insects cause a local itching swelling, very like urticaria and occasionally resembling erythema nodosum. Sometimes the swelling is as great as in angio-neurotic œdema. The bite of a fly may prove fatal by conveying infection and setting up pyæmia. *Wasp stings* are usually trifling, but may produce drowsiness, urticaria, swelling of eyelids and lips, and croupy breathing, with perhaps nausea and vomiting. A single sting has proved fatal in 25 minutes. *Bee stings* are more severe and convey 3 poisons, convulsive, stupefying and inflammatory. The local effects are redness, swelling and itching. Constitutional symptoms are nausea, vomiting, pallor, giddiness, prostration and general urticaria. Alcohol and ammonia must be given.

*Fleas* rarely attack the face. Extensive bites may cause fever, up to 103° F., restlessness, insomnia, and general malnutrition. Erythematous wheals, and even bullæ, may be produced.

*Bugs* constantly attack the face and hairless scalp. They may cause ill-defined, dusky, œdematous blotches, and neither wheals nor irritation.



The puncture often cannot be seen without a lens, and may bleed at times. According to Hutchinson urticaria pigmentosa is due to this cause.

*Preventive treatment* consists in keeping off or catching the insects. Sulphur internally, sulphur soap or chinosol in the bath, and the application to exposed parts of tr. pyrethri, musk, carbolic acid, or the oils of lavender, cloves, citronella, cinnamon and eucalyptus may keep them off. Suitable preventives for fleas and bugs are powdered cloves dusted on the bed-clothes, essence of thyme, and pledgets of absorbent wool sprinkled with 50 per cent. Jeyes fluid and wrapped up in the night garments during the day.

*For relief* of the irritation apply Eau de Cologne or vinegar and water, a raw onion, lead lotion, strong ammonia, carbolic acid 5-10 per cent., menthol 1 part in 4 of olive oil or spirit, or ichthyol collodion.

**Harvest Bugs** (*leptus autumnale* or *bête rouge*) produce the harvest rash, *erythema autumnale* or *prurigo du Rouget*, in the months of July to September. They are minute, almost spherical insects, with 6 legs and sharp mandibles, red or yellowish red in colour, somewhat like cayenne pepper, and extremely active. A lens is required in order to see them. They chiefly attack the legs and ankles of children in hay and cornfields, about French beans and in chalky places. Burrowing into and burying themselves in the epidermis, or in a hair follicle, they give rise to intolerable local itching. A papule or wheal is formed and runs its course in about a week. If the head only penetrates the epidermis, the abdomen swells until visible as a bright red dot. Sometimes there is a general urticaria. The itching causes insomnia and scratching, which leads to impetigo, pustules, ulceration, and hæmorrhage into and round the papules, producing a purplish zone.

The preventive measures suitable in the case of other biting insects should be adopted for children who play about in infected fields. Give a creolin bath at bedtime to destroy the parasite; or apply balsam of Peru or benzene, followed by a warm bath. Use the ordinary remedies for the relief of insect bites, and an ointment of sulphur, carbolic acid, picis liq. or hyd. subchlor.

**Scabies**, due to the *acarus scabiei*, is acquired by direct contact with a pre-existing case or infected garments. The parasites invade the tender, protected parts of the skin, between the fingers and toes, the axillæ, genitals and buttocks. The feet of infants are often affected because they play with them. The male *acarus* lives on the surface, while the female burrows into the horny layer of the skin. She gives rise to great irritation, and a papulo-vesicular or pustular rash, especially on the hands and fingers. Pustulation is common in children. The pustules vary in size and may coalesce. On the trunk and buttocks the rash is very like lichen urticatus. It may be confined to the lumbar region. The burrows are white or black, according to the degree of cleanliness. They may be visible with a lens



between and on the sides of the fingers and toes, and on the sides of the feet, but they are often invisible or indistinct, being easily destroyed by scratching and vesiculation. On scratching away the epidermis at the distal end of a burrow, with a needle on a holder, the parasite clings to the needle and can be examined in liq. potassæ.

At first there may be merely itching, chiefly at night, and perhaps patches of transitory erythema. The scratching and itching soon induce a polymorphic eruption. A limited number of small itching vesicles in characteristic places, with surrounding healthy skin, is not uncommon and is significant, though burrows may not be visible. These are the true acarian vesicles, chronologically the first sign. The face and scalp are never attacked, except in infants. Scabies, caught from dogs, gives rise to a papulo-vesicular rash and no typical burrows. Impetigo, ecthyma, lymphangitis, adenitis and albuminuria are complications. Dermatitis may be due to treatment. Untreated cases vary in duration and severity, according to the state of health, hygienic surroundings and cleanliness, but do not get well spontaneously.

*Treatment.*—Destroy the parasite, prevent re-infection, and cure the rash. Boil or stove all linen, gloves and bedclothes. Give a hot bath, using soft soap and a nail brush, and then rub in sulphur ointment, or balsam of Peru 1 part in 5 of olive oil. The balsam should be painted over the skin and rubbed in gently. Sulphur ointment is too strong for infants. It must not be rubbed in as it is apt to set up dermatitis. Tr. benzoini may be rubbed in twice daily and followed by a bath in 2 days' time. Liq. calcis sulphurata is strong enough for mild cases. Sulphur baths are rarely needed. Both storax and balsam of Peru have been followed by albuminuria; and a fatal case of acute nephritis in a boy, aged 15, has occurred after the use of the balsam, so it is advisable to examine the urine previously. There is no risk, if it is only applied for 6-8 hours and a hot bath then given. Another suitable ointment is  $\beta$ -naphthol, 5-10 per cent.; it neither stains nor smells unpleasant, and does not aggravate secondary rashes. After 2-4 days of the use of ointments give a bath and apply zinc oil, or ac. salicyl. gr. 5, ol. ric. 40, ol. olivæ ad 100 parts, for the dermatitis.

**Pediculosis vel Phtheiriasis** (*the lousy diathesis*).—Pediculi are true insecta. They suck blood by means of a haustellum or proboscis, an elongation of the œsophagus, and do not bite. The male is smaller than the female. The nits are attached by cement substance to the hairs, and have an operculum or lid, and cannot be brushed off. Multiplication is very rapid. *P. Capitis* has a triangular head. *P. Vestimentorum* is the largest, and has a more oval head and narrower thorax. *P. Pubis* is the broadest and has a crab-like body; and the thorax is not differentiated from the abdominal segment as it is in the other varieties. It is rare but may affect the eyelashes, and is then readily destroyed by ung. hyd. oxid. flav.



The body louse is found where the clothes are tight. It causes reflex shivery movements, and pigmentation and scarring due to scratching. Nits are found on the short hairs. Give a sulphur or carbolic acid bath, and rub ung. staphysagriæ into neck bands, etc. Alkaline baths relieve irritation.

The head louse is far the most common. It produces no immediate effects, but the scratching and irritation lead to secondary infection, suppurative and ulcerative affections of the scalp, and secondary adenitis. The vermin must be destroyed and the nits removed. Local applications must be free from danger, non-injurious to the hair or scalp, destructive of nits, certain and rapid in action, and inexpensive. Soak the head with oil of sassafras and cover it with a calico cap for 8-12 hours. Comb with a fine comb and shampoo well. This destroys both vermin and nits, and a pint bottle, price 3s. 6d., is enough for 30 children. Other suitable methods are hot carbolic lotion, 1 in 40, acetic acid, and vinegar, alone or with 0.5 per cent. hyd. perchlor. Petroleum, paraffin and naphtha 5 per cent. are dangerous in the presence of a light. Acetic acid dissolves the cement and the nits can then be combed off. Greasy applications, e.g. hyd. amm. gr. 10, ol. olivæ m. 10, lard oz. 1, kill the parasite by blocking up the air passages. An elegant and quick method is to use 1-1½ pints of methylated or rectified spirit, pouring it from a jug slowly from behind, with the hair thrown forward and the head over a basin. Then comb with a wide toothed comb or brush well. One application is sufficient. The hair is then washed with a little milk and exposed to sun, to restore its lustre. The spirit causes smarting if the skin is inflamed or broken, is expensive, and must not be used near a naked light. Equal parts of xylol and spirit or ether, rubbed in with a mop of absorbent wool, is rapid and efficacious. The nits can be easily combed off.

**Creeping Larvæ.**—*Syn.*: *Hyponoderma*.—A creeping eruption of the skin has been described and named *myiasis linearis* or *larva migrans*, but no larva or parasite has been discovered. It occurs chiefly in Russia and Arabia. Lee (1870) reported a case in a girl, aged 3 years, the eruption beginning below the ankle and travelling up the thigh and on to the abdomen. In a girl, 4 years old (Van Harlingen, 1902), the eruption began on the foot as a line of small vesicles with a peculiar beaded look, the vesicles containing black granules; and in a boy, 5 years old, it began on the sole and extended on to the dorsum as a serpiginous bright red rash, painful and itchy, and feeling like whipcord under the skin. Crocker has noted 7 inches progress in one day. Thus, its main characters are the progress, serpiginous aspect, and sometimes itching. It should be treated by tar ointment, electrolysis, or incision and curettage at the end of the track of the eruption.







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